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IOWA STATE COLLEGE OF AGRICULTURE
AND MECHANIC ARTS

Vol. XVI. NOVEMBER 7, 1917 No. 23

REPORT OF

PRACTITIONER'S SHORT COURSE IN

VETERINARY MEDICINE

IOWA STATE COLLEGE OF AGRICULTURE
AND MECHANIC ARTS

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Vol. II No. 1

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REPORT OF PRACTITIONER'S SHORT COURSE IOWA STATE COLLEGE OF AGRICULTURE AND MECHANIC ARTS

VOL. II

SEPTEMBER 4-9, 1916

No. 1

DIVISION OF VETERINARY MEDICINE

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*First date after the name indicates date of appointment to professional position, the second date, when the first fails to do so, indicates the date of first appointment in the College.

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Thomas Seeter Leith, D. V. M., *Anatomy*, 1916.
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B. Agr., University of Wisconsin, 1885; M. S., 1889; Ph. D., Washington University, St. Louis, 1898.

William Harper Pew, 1912, 1909, *Professor of Animal Husbandry,*
B. S. A., Iowa State College, 1907.

Henry Elijah Summers, 1898, *Professor of Zoology.*
B. S., Cornell University, 1886.

Lester S. Gillette, 1915, 1914, *Associate Professor of Animal Husbandry in Charge of Dairy Farm.*
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B. S. A., Iowa State College, 1909.

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B. S., University of Iowa, 1901; D. D. S., 1903.

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J. C. Bowman, 1914, 1910, *Associate Professor of English.*

J. O. Rankin, 1916, *Associate Professor of Economic Science.*
A. B., Tarkio College, 1904; B. Sc., Iowa State College, 1908; M. A., George Washington University, 1912.

George Judisch, *Veterinary Pharmacology*, 1912, 1901.

Henry Hugh Dukes, B. S., 1915, *Physiology.*

Earl A. Hewitt, A. B., B. S., 1915, *Anatomy.*

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PRACTITIONERS' SHORT COURSE IN VETERINARY MEDICINE.

Iowa State College of Agriculture and Mechanic Arts.
Ames, Iowa. September 4-9, 1916.

DAY	8:00—10:00	10:00—12:00	1:00—3:00	3:00—5:00
Monday	Registration	Pathology of Infection Dr. W. W. Dimock	Further studies of Teeth and Sinuses of the Horse Dr. H. S. Murphey	Surgery of Nasal Cavity and Sinuses Drs. Bemis and Guard
Tuesday	Anatomy of Foot Drs. Murphey and Grossman	Physiology of Digestion in Herbivora Dr. H. D. Bergman	Physical Exam. of Digestive Tract and Indigestion in Cattle Drs. White and Bolton	Rumenotomy Drs. Nelson and Bemis
Wednesday	Anatomy of Foot Drs. Murphey and Grossman	Diseases of Foot Dr. J. W. Adams	Surgery of the Foot. Operations for quittor sidebone, penetrating street nail Drs. Adams and Bemis	
Thursday	Hemorrhagic Septicemia, Serum and Vac. Immunization Dr. Chas. Murray	Shoeing and Balancing Dr. J. W. Adams	Physical Exam. of Resp. Tract and Pneumonias Drs. White and Bolton	Thoracentesis Dr. R. R. Bolton
Friday	Parasites common to Domestic Animals of Iowa Dr. W. W. Dimock	Therapeutic measures in eradication of parasites Dr. H. D. Bergman	Influenza and Shipping Fever Dr. D. S. White	Post mortem demonstration and preparation of Tissues for Shipment to Laboratory
Saturday	Special Clinical Features			Drs. Dimock and Willey

Evening program starting at 8:00 p. m. Tuesday: Address—President R. A. Pearson.
Wednesday: Problems of the Profession—Dr. D. S. White.
Thursday: Poisonous Plants—Dr. L. H. Pammel.

ADDRESS OF WELCOME

PRESIDENT R. A. PEARSON

President Pearson was introduced by Dr. Bemis, who in introducing him, said:

I have the honor to introduce to you the speaker of the evening. I assure you that the introduction will be very short, but I do want to say that I am sure you will be glad to meet him because he is the President of this College, and because we feel that he is a college president who is in very close touch with the work of the veterinary profession, both because of his knowledge of the very eminent services to our profession of his brother, Dr. Leonard Pearson, and also by reason of the work of the speaker as Commissioner of Agriculture of the State of New York, during which time he tells me that his association and relations with the members of the veterinary profession were very close.

And so we feel that we have a college president who appreciates the work that the veterinary profession is attempting to do. I am very glad to introduce President Pearson. (Applause.)

Dr. Bemis and Gentlemen: I am very glad to appear and say a few words of welcome. It seems that the address of welcome has to come on the program somewhere between the beginning and the end. I believe that actions speak louder than words, and I think you have judged already whether you are welcome, by the evidences that have been surrounding you ever since you arrived.

But I am glad to say this formal word of welcome. I appreciate the kind introduction that Dr. Bemis has given me. I have been associated more or less with veterinarians for four strenuous years in New York, rather closely associated with them, and I have the idea that that was one of the principal reasons why I was considered good enough to be promoted to the State of Iowa. So I really feel that I owe a good deal to the veterinary profession.

I want to say a word or two about this institution. This is pre-eminently the practical and the technical institution of higher learning in the State of Iowa. It is the land grant institution of this State. Here we are proceeding along five lines of technical or constructive work. We have our work divided into these five branches: Agriculture, Engineering, Industrial Science, Home Economics for the girls and Veterinary Medicine, and in these five ways this College is endeavoring to

serve the State of Iowa and in each line we are endeavoring to serve them in respect to investigation, college teaching and extension work.

I suppose that the scientists engaged in these different lines of work and in the other lines of work could hardly show as rapid advancement as has been made in the veterinary profession in recent years. It is said that you may judge of the progress of a profession by looking at its library. It is not very many years ago since any one of us could have walked into a good veterinary library and have carried it all away on one arm. Today we would have to call an express wagon to carry it away, and then we might have some difficulty in getting all the books on one wagon.

The advancement of the veterinary profession has been indicated perhaps as well through our government's activities as in any other one way. Do you appreciate that it was only in eighteen hundred and seventy something, when most of us were just starting our earthly career, that our United States Government gave its first real official recognition to the veterinary profession? In the Treasury Department at that time a cattle commission was organized for the purpose of combating and controlling and eradicating contagious pleuropneumonia which had found its way into this country. Since then our government's activities have gone forward very rapidly, and you know better than I what they are now, and the enormous out-reaching efforts of the Federal Bureau of Animal Industry, and gradually our veterinarians are being recognized even beyond that.

I want to congratulate you upon the recognition you have received through the new army bill. At last veterinarians have something that has long been due them in reference to rank and responsibility in the United States army. I suppose my old friend, Dr. Hoskins, of Philadelphia, has had that bill under his wing for a good many years. He is one of the men principally responsible for its success. But certainly veterinarians everywhere have been interested in it and have been back of it.

The veterinary industry relates itself, of course, to some enormous interests in this country, and I might have taken the trouble to have looked up the exact figures and given them to you, but I expect you have already heard them many times and have forgotten them. It is impossible for us to appreciate the enormousness of some things. It is impossible for us to appreciate the enormousness of the live stock interests of this country.

A great astronomer recently was trying to tell a few of us how far it is from this earth to a star, and he did it in this

way. He said: "If I should tell you the number of billions or trillions of miles to that star you would not remember it, but," he said, "I will give you an illustration which will help you to remember it. There is a certain spider which spins a very fine web. It is supposed to be the finest threadlike thing that is known in nature. So fine," he said, "that the manufacturers of the best microscopes and telescopes use the thread of that spider's web to go across their lenses so as to mark the field. That web is so fine that if the spider were able to do so and could spin one thread all the way around this world and come back to the place where it started it would take only a pound of the web to span that distance. Now," he said, "if that spider should start from this world and climb up to one of the nearby stars, not a distant one but one of the nearby stars, it would take as much of its web as could be carried on a fifty-car freight train on the Pennsylvania Road." And he said, "If that spider should go on to one of the most distant stars it would take as much of its web as could be carried on a thousand such trains. Now you know how far it is to a star, do you not?"

But, suffice it to say that the enormousness of the live stock interests of the country comes so close to the human interests of this country that they affect directly or indirectly every human being. They affect us through their enormous commercial interests and possibilities, and they affect us through our food supplies, and they affect us through our health, for we are all of us laymen coming to know that there are some very important diseases easily transmitted from animals to men.

Good veterinarians are needed, and are needed very much. Some persons think that with the coming of the automobile and the fast railroad trains the need for veterinarians is decreasing, but the contrary is true. If this country had to choose today which it would have for the future, its live stock or automobiles and railroad trains, there is no question how that choice would fall. The need of the veterinarian is greater today than ever before, and it is becoming still greater, I mean for the good veterinarian.

The engineers who devise the railroad trains and the automobiles are never going to crowd out the veterinarians. I have heard that there is a good deal of conflict between the engineers and certain other classes here. I have been told that engineering instruction and agricultural instruction cannot thrive at the same college because they are so incompatible, and I looked into that subject with considerable care, and I have found them incompatible in only one way. Our agricultural faculty here is trying the best it can to increase the crop yield in the State of Iowa. They are trying to produce

two tons of hay where only one ton grew before, and fifty bushels of corn where only twenty-five grew before, and in that way they are trying to increase the size of the State of Iowa, and they are succeeding pretty well. We expect that if their work continues as it has been, this State will be four times as large as it is now, in the not very distant future, because it will be raising four times as much of a crop in the food line. On the other hand, our engineers are putting on straighter railroad tracks, and more telephone lines and telegraph lines, and in that way they are bringing distant points closer together, and they are making the State shrink up from year to year, and that is the only conflict that I know of between the engineering and agricultural departments. There is no more conflict between the engineering and the veterinary business.

I say we need more good practitioners. It is evident that we do, because we are told that the financial losses from just a few of the principal live stock diseases amount in a single year to the enormous sum of $300,000,000 or more. I have been figuring it. Supposing that the veterinarians would come forward with a proposition to the farmer and should say, "We will go fifty-fifty with you on that." That would mean that the veterinarian would have $150,000,000 and the farmers would save $150,000,000. I have such confidence in the veterinarians that I believe they could save practically all of that if they had it their own way. If the veterinarians were going to save $150,000,000 it would be fair, I assume, to say that there should be 15,000 additional veterinarians. That is allowing only $10,000 a year for a man, which I understand is a small income for a good veterinarian. (Laughter.)

So there is proof, gentlemen, that we need more good veterinarians. We want veterinarians to save that $300,000,000, and then some more hundreds of millions in the other diseases which I have not referred to.

Some communities accept these live stock diseases as a matter of course. They stand them as they would a tax only much more cheerfully than a tax. They scold about a tax, but they take the losses from these diseases as good naturedly as can be. The State of Iowa suffered $20,000,000 loss from hog cholera three or four years ago, and I did not hear very many protests on that subject. If the Legislature had attempted to tax the State $1,000,000 every year for some worthy purpose, we would have heard a good many protests, I am sure.

It reminds me of a certain ignorant gentleman who was infested in a certain kind of way. He developed a cold on his lungs, and went to a doctor to be treated, and the doctor

looked him over. The patient had put on a clean, white-bosomed shirt. In the course of the examination the doctor rapped his bosom swiftly to see what the sound would be, and a little volume of smoke came out from the crease in his shirt, and the doctor drew back and said, "What is that?" The man said ,"That's insect powder. Don't you use it?" (Laughter.)

Some of our communities have accepted the presence of animal diseases with the same apparent degree of satisfaction that that ignorant person had accepted his unnecessary infliction, I am sorry to say. I do not want to reflect upon the intelligence of the good people of Iowa. I direct those remarks more particularly to some other State, not Pennsylvania nor yet Ohio, but to some other State (laughter); to some States farther south and west.

Now, seriously, we ought not to permit these losses to continue. If I have any hobby at all it is in the reasonable proper use of our natural resources. I consider that it is nothing short of an outrage to destroy our coal supply as we are doing, and use up and waste our iron as we are doing, and it is nothing less than an outrage to permit these animal diseases to go on with their ravages as they are doing, causing the public to carry these enormous burdens of loss. And, gentlemen, it is up to the veterinarians to save us, and I have shown you how you can make a lot of money by doing it.

We know that the veterinary field is opening up constantly as well for the workers in other phases of veterinary and sanitary science. Not only is the United States Government calling for them, but State governments are calling for them more and more. And now the cities and towns are demanding them. I suppose you have thought many times what the phrase "The Federal inspection of meat" is, so far as our local communities are concerned after all, when that represents only a small proportion of the meat that is consumed locally. And our United States Government is spending something like $3,000,000 or more a year on meat inspection, and that represents only a very small part which comes into our cities and towns at home, and the balance comes from uninspected abattoirs. We all know that animals that could not pass the inspection at the government plant are often sent to other places where no veterinarian sees them. Fortunately, the cities and states are waking up to the fact, and are seeking the seriousness of its bearing on public health and its indecency, and they are looking more and more to the veterinarians to help them out of those wretched conditions.

Then, too, we find that stock farms and breeders are depending more and more upon the veterinarians, and I notice that the veterinarians themselves are getting into that line of

work, and I can hardly think of a more useful line for a man to take up, or a better preparation than animal husbandry and the veterinary training combined, if he were going into stock farm work.

We laymen are coming to see also that veterinary science is as difficult as any science to understand. The mucous membrane of a cow is just as complicated as the mucous membrane of a human being, and the diseases are just as difficult to diagnose and treat. It has been said that the veterinary work is more difficult than the work of the human practitioner, because he has more animals to contend with. Somebody says the physician has four kinds: husbands, wives, bachelors and old maids and the veterinarians have seven or eight different kinds which you have to be familiar with.

Now, the word veterinarian is coming to mean something worth while in our great country. In some sections it has not been known until lately and perhaps in some sections it is not known yet. They say that a couple of gentlemen were driving down through South Carolina one time and their horse met with an injury and they had to tie him to a tree and walk in town. They asked a citizen of the place if there was a veterinarian there, and he said he did not know. He said, "We have Baptists and Methodists, but I don't know of any veterinarians here." (Laughter.)

I think that the word veterinarian is coming to be recognized by intelligent people as a synonym for a certain very peculiar and honorable kind of skill, and I like particularly to emphasize the word "honorable" because I believe that the veterinary profession, owing to its newness has suffered more or less in respect to the honorable side.

While I was in office in New York State it happened to be my duty to enforce the agricultural law, which included the law relating to animal diseases, and there are some severe penalties in connection with improper practices. At one time it became my duty on the advice of our chief veterinarian and attorney, to withdraw the license to practice from a veterinary surgeon because of some highly dishonorable things which he had done. And within two weeks after that I heard that that gentleman had gone into human practice and hung out his sign to that effect. So I thought that in that section of the State at least we could say that the veterinary profession was honorable as compared with some of our highly honorable professions.

Now, gentlemen, these are great days for searching out and revealing the truth. In the various States through our agricultural experiment stations we are spending now over $5,000,000 a year in the search for truth, including veterinary

truth, and agricultural truth, and an enormous amount of money in addition is being expended under the United States Government in the search for truth. How our troubles would fade away if we only could know the truth.

I recall the story of a lynching bee that had their victim all but hung up to a tree and the excitement, of course, was running high and there was a great throng of men determined on carrying out their purpose and suddenly there came a man rushing in from the side of the crowd, accompanied by the victim and said, "He is not guilty," and that lynching bee quieted down at once and faded away. They had the truth.

Now, that is a radical occurrence, but to a lesser degree, in the same way, the state of our mind will settle down and we will become thoroughly satisfied, when we have been excited and uncertain and perhaps disagreeable because we did not know the truth.

How many mothers are there throughout this country tonight who would give everything they have if they could only know the truth about their boy who is miles and miles away. I know what is running through the minds of those mothers.

And the Government needs truth. How desperately our Congress needed it a few days ago when they were dealing with this railroad strike situation. If by some stroke of Providence the truth could have been dropped down upon that Congress they certainly would have acted wisely.

And the physician, all he needs to know is the truth as he looks upon his patient; if he only could know what is taking place within that body and why it is taking place and what would remedy that difficulty, the whole trouble would be over at once.

And the judge upon the bench is only looking for the truth, and when he knows the truth he knows how to dispense real justice. And the people are looking for the truth; they want it.

Speaking of New York State—Dr. Bemis got me into this —I remember one time one of our veterinarians came in looking much the worse for wear. He said that in a little community up near Utica he had almost been thrown out of a hall because he had tried to tell those people something about tuberculin. They had had some bad luck with it and they were prejudiced against it and they did not want to hear anything about it, and they broke up the meeting and he was glad to get away with a whole skin. Well, we talked the thing over and we decided that the only thing those people needed was the truth and we gave it to them, and we gave them a big

dose of it. So we had a carload of reactors shipped in there. It was announced through the papers that they would arrive on a certain day and they were going to be displayed in the market place, and every one was invited to come in and see them. So these veterinarians went up there, I and a few others did and I walked around among the crowd and heard some very uncomplimentary remarks about myself. But I was well satisfied because I felt that those men were going to know the truth before they got away from there.

Along about ten or eleven o'clock it was my privilege to get up on top of a barrel and call about five or six hundred farmers to order, and I told them that every one of those animals had reacted to the tuberculin test, and we were going to show them whether tuberculin amounted to anything or not, and I asked them to appoint a committee of three to pick out the animals that they thought were the healthiest and we would see if they had tuberculosis. And they did. They picked out a fine little black heifer, the picture of health, and I felt a little bit uneasy, I will admit. The butcher knocked her down and the veterinarian began to examine her, and her lungs were as nice as could be and the lympathics were just perfect, and they opened up her abdomen and everything was all right down there and I would have given a five dollar bill if they had started on another animal. And then they opened up the udder and about half a pint of pus came out and the people who were standing by were appalled. The truth hit them just like that and the excitement began to subside.

We got out another one, selected by the committee, a fine, healthy looking animal, and in a few minutes the veterinarian held up the tuberculous organs and there they were. You could not get away from that. They were the truth. The people saw it and heard it themselves, and they had to recognize it. And they killed and examined a third one and the same thing occurred. Then I asked them if they wanted to kill them all or would they like to pick out one that they thought had it the worst and they seemed satisfied to do that. They brought out an old cow with about fifteen wrinkles on her horns and I knew that that was a good one for my case. They brought her up and killed her and she sure did have it bad. She looked just like it, everybody thought so, and they killed her and they looked for a long time before they found some of the smallest tubercles that I have ever found in any animal.

Then we went into the hall and our veterinarian gave them such a talk on tuberculosis and tuberculin as they had never heard before and when I arose to say that the meeting was dismissed, some husky farmer with his pants in his boots

came up in front and said: "Hold on, Mr. Man. I want to say something to you. We had a doctor up here the other day who talked to us about tuberculin and we did not treat him very well and we have had him come back here today and they have shown us something," and he said, "I believe that tuberculin is a good thing and I believe that every man in this hall thinks so, too, and so as to show the Commissioner here that we think it is good and should be used, all those who think it is, stand up." And every man in the hall stood up on his feet. It was just because they had the truth and they were looking for the truth.

And now I am coming to the point of my address and that is my stopping place.

Veterinary science is developing very rapidly. I referred to the libraries. Here in Dr. Bemis' office there is not a day goes by but what pamphlets come in, new pamphlets, new discoveries, new investigations, new truths that have been developed by painstaking men, here and there, and who are working day and night in search of truth. It is coming on us all the time. Here at this one little point in this State, the State has established an institution to gather together all of those bits of truth that are flying about everywhere, so fast that no one of us can keep track of them now. And you busy practitioners who are obliged to go to your work from day to day and have not time to sit down in a laboratory and study out these truths for yourselves and who must depend upon the work of others, you are invited to come in here from time to time under this new plan for the practitioner's course, and hear the newest and latest truths in veterinary science.

Now, gentlemen, we are sorry that our Dean, Dr. Stange, is not here tonight, a man who is known to all of you, I presume. He has overworked and he has had to go away. He is getting a good rest and we are confidently hoping that he will be back here in the near future, fully restored to health, but we are glad that the other members of our faculty are here to help you, each one along his own special line, and we consider ourselves very fortunate, indeed, that we can bring in such able men as Dr. Adams, from Pennsylvania, and Dean White, of Ohio, to assist us in presenting the program to you and the other speakers who will come along from time to time.

I used to know Dr. Adams years ago. I almost think he and I were boys together. He tells me that he originated up in Minnesota, and I told him that long association with the Quakers in Philadelphia has not spoiled him very much. We think it is a great thing to have him come out to Iowa and assist us along his special line, because he is one of the men who was the first to recognize some of these new truths. I

was visiting the State Veterinary College in New York only a week or so ago, and Dean Moore told me of the splendid assistance that Dr. Adams had given them there, and I was glad to tell the Dean that he was coming out here to help us in the same way, and that we are equally glad to have the other strong help which is provided for on this program.

I think that the future of the veterinary profession is very bright, indeed. I think we can look into the not distant future and see a little more of it than we can see at just this minute. We know this, that we are great followers in this country of the people in European countries who have had to face and solve a good many problems some years before we were forced to do the same thing. In agriculture they were the first to develop under-drainage, and they had it well developed when we began to dabble in it a little bit, and our best ideas of under-drainage have followed along with their best ideas, and we have been a little bit behind them in most of this development.

And when it came to using commercial fertilizers they were the first to see the need of them, and they adapted them to their uses, and later we followed their example.

So it was with the establishment of our agricultural colleges. Now, I think we can safely say that we are following just as safely in their footsteps in our veterinary development. Over there they have had veterinary colleges for more than a hundred years. The veterinarians in those countries are looked upon as among the most learned scientists, and the mostly highly respected of all their professional men. And we can see the same thing in this country, when our veterinarians will have the satisfaction of feeling that their great work is appreciated by the public and they are standing on the same level with other scientists engaged in other lines. And, gentlemen, the thing that will enable us to do that is the acquirement of the truth, which we are assembled here to secure.

I certainly wish you well in this short course. (Applause.)

THE PATHOLOGY OF INFECTION

DR. W. W. DIMOCK, AMES, IOWA

In our consideration of the Pathology of Infection, or those morbid and reactive processes which result from the presence and action of injurious agents that may be classed as infectious, it will be necessary to limit the discussion to some of the more common and general factors that logically come within the scope of this problem. Therefore, the following headings are used with the idea that it will simplify and clarify the question, as a whole, and enable us readily and correctly to interpret the conclusions that are naturally our objective:

1. Infection.
2. Specific infection.
3. Inflammation.
4. Wound infection.
5. Local infection.
6. Fever.
7. Secondary infection.
8. Mixed infection.
9. Secondary localization of infection, both specific and non-specific.

By infection is meant the entrance into the animal body of a living micro-organism, its multiplication and as a result of this growth and activity, the production of disease.

By specific infection is understood an infection the same as above, but by a particular species of pathogenic micro-organism. The various specific infectious diseases of animals are caused by specific infections. The lesions resulting from infections are in general more or less the same and often characteristic. They may, however, be local or general, and may frequently vary in different tissues and different individuals. If a particular species of pathogenic organism is present the disease produced is a specific infectious disease regardless of the character of the lesion; on the other hand, regardless of the similarity of the lesions, either gross or microscopic, unless the specific etiological factor is present, it cannot be that disease. (See note below.)

Before taking up the tissue or body reaction to infection, it will be well at least to enumerate the portals of entrance.

1. Alimentary tract.
2. Respiratory tract.
3. Eye and ear.
4. Urogenital tract.
5. Wounds of both skin and mucous membranes.

6. Bites of insects.
7. Through the skin.
8. From loss of tone and lowered resistance of the tissues through disease, lack of nutrition and altered function.
9. Secondary to or along with other infections.

Inflammation

By inflammation we understand the reaction that takes place in a living tissue following irritation or injury by some injurious agent (actual or referred).

NOTE.—To better illustrate the importance and necessity of identifying lesions when possible according to the etiology rather than wholly from the character of the lesion, I wish to call your attention to some specimens that I have here.

Many times in holding post-mortem examinations on animals we find areas of degeneration or necrosis and not unfrequently calcification. Such lesions are often whitish or grayish in color, and one of the first things we think of is tuberculosis, provided of course we are dealing with a species in which this disease occurs. It makes no difference how closely these different lesions may resemble one another, if the etiological factor (example, bacillus tuberculosis), is not there, then it cannot be tuberculosis. On the other hand, if the tubercle bacillus is there, then, regardless of how much the lesions in color and character may vary, from what is considered typical of tuberculosis, it is tubercular. For example, here is the heart from a cow. This heart came from a fat three-year-old heifer. I believe the history was this animal failed to breed, and was fattened and killed for beef, when the heart was taken out it was seen that there was something abnormal. A veterinarian was called and suspecting tuberculosis, and realizing that it was an unusual specimen, shipped it to the Department of Pathology for confirmation of his diagnosis and for use as a museum specimen. The wall of the heart, as you can see, is apparently thinner than normal, and the grayish white material completely surrounding the heart, at least two inches in thickness, is the result of a chronic productive inflammation. There is not much evidence of necrosis in the muscular portion of the heart that can be seen by the naked eye. However, there are many small tubercles on the endocardium and organisms were found all through the inflammatory exudate and in the nodules so that we know it is tubercular. Compare this with this lymph gland which shows a central necrotic mass surrounded by a capsule which is greatly thickened. There is not much resemblance between it and the heart, but they are both tubercular because they are primarily the result of the action of the bacillus of tuberculosis.

I have here, another specimen in which the contrast is still greater, the one on the right is a piece of omentum, covered with nodules, which are plainly tubercular in character, the other is a piece of omentum which was put in to bring out the difference in gross appearance of etiologically the same condition. On the surface of the latter we have a very delicate fibrinous exudate. The exudate is rather firmly attached, probably from the beginning of fibrous connective tissue formation. In smears from this delicate exudate we found the tubercular organism.

The great difference in appearance of lesions, etiologically the same, either from age, location, complications or species of animal in which found, and the similarity of lesions when brought about by an entirely different and distinct cause emphasizes the necessity of classifying lesions and diseases so far as possible according to their etiology.

In studying inflammation it is convenient to consider the following points:

1. The cause.
2. The location or tissue involved.
3. The damage or injury to the tissue.
4. The reaction.
   (a) To the injury.
   (b) To the injurious agent.

The Cause—While various agents other than micro-organisms may be the cause of inflammation, only bacteria and the toxic substances which they produce will be considered since they are the most common cause.

Bacteria may be directly responsible for starting the inflammatory reaction.

Bacteria may act jointly with trauma, accidental or operative wounds, or their invasion of the injured structure may be entirely secondary. In this latter case the bacteria may come from without, or bacteria already in the system may localize in the injured part. (I shall emphasize this point later.)

It is important to know the cause of the inflammation because, from cases of experimental and natural infection, we have come to know that certain organisms are possessed of rather definite biological properties; gain entrance to the body through certain channels and under certain conditions; are limited in their manner of action and distribution in the body; produce a rather definite type of injury; and stimulate to activity certain elements in the body, resulting in a rather definite type of reaction, constituting as a whole, in many instances, a rather characteristic lesion.

The action of the injurious agent may be mild or severe;

may take place at once and be self-limiting, or may be continued over a long period; again the infection may be present for a long time, in some cases for months, before gross lesions appear. From a knowledge of the general facts stated above, the pathological and clinical manifestations become interpretable.

The location or tissue involved is important because of the difference in tissues to withstand the injury, their power of regeneration, and the proximity of the affected area to less resistant structures and vital parts. Often an insignificant injury to a very resistant structure may, if not properly cared for, become serious from extension and involvement of other parts.

By the injury or damage we are to understand the tissue changes retrograde in character, that result from the action of the injurious agent be it from trauma, infection or both. When we speak of the lesion, it includes both the changes that result from the harmful agent or agents, and the changes that constitute the reaction. In other words, it includes all the tissue changes, local or general, regardless of the origin.

The changes resulting from the irritant alone may not be evident morphologically. For example, in cases of tetanus the changes being largely functional there are very few demonstrable lesions, except at the point of inoculation, and in many cases even that is never found. In other instances, the damage resulting from the action of the injurious agent is very marked and evident. Take, for example, the disintegration of tissue from the action of the bacillus necrophorus. Cells may die at once and show no morphological change, or, rather marked morphological changes may be evident for some time before the cells actually die.

The damage to the tissue from the infections that cause inflammation are, possibly first, in some instances, to a degree mechanical. More commonly, however, degeneration and necrosis of individual cells or groups of cells and in some cases, complete destruction of all the elements, even the most resistant as connective tissues, is the result of the action of bacterial toxins. Other causes of retrograde changes are interference with nutrition, due, no doubt, to structural and chemical changes in the area; to circulatory disturbances and to toxic substances from the necrotic cells and broken down tissue elements.

In a general way we can separate the cells of the body into two groups, the parenchymatous structures which include the more highly specialized cells, for example, the kidney and nerve cells which perform rather special functions, and the connective tissue cells or supporting structures of the animal

body. An injurious agent that will kill a parenchymatous cell may only stimulate the connective tissue cell to reproduce. The first action of irritating toxins is, if strong, to kill the cell; next, to start degeneration, and last, if mild, to stimulate the cell to its maximum physiologic activity and often to abnormal activity. The first structural change is degeneration usually beginning with cloudy swelling. Other and more serious forms of degeneration may follow, or be primary, any or all of which may terminate in death of the cell. The cells in the center of the infected area die first; those just outside are in a state of degeneration and will probably die; while those still farther away are affected but will not necessarily die; while those that border on the involved area are stimulated to increased activity through the action of the diluted toxin that has reached them.

This brings us to the point where the reaction on the part of the tissue and body as a whole is to be taken into consideration. Under reaction it should be remembered that the cause of the reaction lies not alone in the injurious agent, but also in the injury and in the toxic irritating substances from the necrosed tissue. The purpose of the reaction is to get rid of, or neutralize the injurious agent, and to repair the damaged area. The reaction is, in general, the same for all injurious agents, yet the fact that for different agents different elements are found, brings up the question of chemotaxis, namely certain infections or irritants are chemotactic for a certain kind of cell (pyogenic cocci for polymorphonuclear leucoytes), or we can explain the presence of certain elements, fluid and cells, in inflammatory areas in this way. The body has at its disposal certain elements which it can use in its effort to check the growth and action of infections and in bringing about recovery. In some cases only one element may be used (serum), or two or more, serum and cells (lymphocytes endothelial and polymorphonuclear leucoytes, etc.). In this connection, we should not ignore the importance of chemical changes that may occur, which are, however, not well understood. This would seem to explain the reason why it became customary to classify inflammation by the character of the exudate.

1. Serous inflammation.
2. Fibrinous inflammation.
3. Purulent inflammation.
4. Caseous inflammation.
5. Hemorrhagic inflammation.
6. Catarrhal inflammation.
7. Necrotic inflammation.
8. Diphtheritic inflammation.
9. Ulcerative inflammation.
10. Productive inflammation.

It is interesting to speculate on theory: Is the character of the exudate governed wholly by the chemotactic properties of the injurious agent, or does the body send out the element, or elements, to combat the injurious agent that its nature and location would seem to demand?

The reaction on the part of the tissue to overcome the injury and the injurious agent constitutes a series of changes, varying materially in different cases, yet all apparently acting to the same end. Circulatory changes, inflammatory exudation, cellular activity, and regeneration constitute the reaction.

Circulatory disturbances are dilitation and alteration of the vessel wall, a slowing of the blood current and changes in the number, position and proportion of cellular elements.

Inflammatory exudation: The principle exudative process is the exudation of fluid and cells. The fluid (lymph) tends to dilute toxins and has some bactericidal action through alexins and lysins in the blood. The formation of fibrin in the lymph exudate is no doubt chemical. It is believed that the disintegration of cells, especially leucocytes, aids in the formation and action of a fibrin ferment on fibrinogen in the fluid exudates. Fibrin may predominate in the lesion, or it may only be recognized on microscopic study in the form of fine threads. While fibrin is one of the most difficult elements in resorption, it evidently is sometimes beneficial in its action. The emigration of leucocytes and diapedesis of red blood cells are often the most evident and important features of the exudation. The polymorphonuclear leucocytes are usually the ones to emigrate first. They mechanically and physiologically destroy or digest micro-organisms and fragments of tissue elements but their principal action is chemical through the action of a substance, enzyme or ferment, which they produce. This chemical substance will neutralize toxins and dissolve broken down tissue elements so that they can be carried away by the blood. Endothelial leucocytes are equally important but are more frequently active in mild chronic processes or follow after the polymorphonuclear. They are decidedly phagocytic in action. The first ones present are through emigration but they multiply in the area and therefore often become the predominating cell.

In certain inflammations lymphocytes predominate and like the other cells, they neutralize and otherwise destroy injurious elements. The other cells normally found in the blood are less frequently seen yet they emigrate in certain cases, for example, an eosinophilia is considered favorable in cases of pneumonia. They are increased in some cases of parasitisms.

All of the above described changes, the injury to the tissues and the reaction (as stated above), constitute, or go to make

up, a lesion. They also explain the long recognized cardinal symptoms of inflammation:

Redness—From increased blood. (Congestion.)

Heat—Also due to increased blood supply and, no doubt, to the increased activity of the affected area both destructive and reparative processes. However, there is some experimental evidence to show that no heat is formed in the part.

Swelling—The swelling is partly due to congestion but largely the result of exudation. (Distension from fluid and cells.)

Pain—The pain results from the pressure on the nerves and probably from irritation.

To the above signs it has become customary to add a fifth symptom of inflammation, namely, altered function.

While we recognize and emphasize the extent and importance of the damage or injury produced by the various injurious agents, it is true that we more often estimate the importance and extent of the damage from the degree and character of the reaction probably to the same degree as we classify the various inflammations, pathologically and clinically, according to the character of the exudate.

Wound Infection

By wound infection we understand those cases of infection where micro-organisms gain entrance to the tissues of the body through interruptions in the continuity of the tissue, accidental traumatisms and operation incisions. The source of the infectious agent in cases of wound infection should receive brief mention. The skin and mucous membrane normally harbor bacteria that, under favorable conditions, are capable of causing disease. The bacteria may reach the injured area along with the object causing the traumatism, from dirt and dust and by flies. In many instances micro-organisms already in the system, but prevented from developing because of the natural protective forces of the body, may be carried to an injured area where they readily become lodged and find there conditions favorable for their growth and development.

The first and most common form of wound infection is met with in those cases where bacteria have gained entrance to the tissues following abrasions and produce local inflammatory processes. The process may be acute or chronic, and the lesions, while varying materially in different cases, comes within the changes met with in acute or chronic inflammations. In some cases, degeneration or suppuration may be marked; on the other hand, evidence of degenerative changes may be absent, the lesions being made up of those processes that result from the inflammatory protective reaction on the part of the

tissue. In the great majority of wound infections, healing takes place by granulation. Conditions such as botryomycosis navel ill, lip and leg ulceration and necrotic stomatatis in many cases, fail to show lesions following the introduction of the infection, but later develop extensive and often very characteristic lesions. It is quite possible in many of the specific conditions which are apparently the result of wounds, that the local reaction is complicated from the presence of a secondary invader. In the cases of botryomycosis the lesion, in the great majority of instances, is purely local and chronic, while in other cases (example, tetanus) no local reaction is evident but the organism exerts its influence on some distant structure in the body. Non-specific micro-organisms may gain entrance to the tissues through wounds, and fail to produce any local reaction, but, in many instances, they are carried by the blood to other structures, where, conditions being favorable, they produce more or less serious lesions. The tissue changes in wound infection, as stated above, all come within the pathological processes dealt with under inflammation. In some cases, there is a simple inflammatory reaction followed by healing by granulation, while in other instances, inflammatory reaction is complicated; that is, all the tissue changes known in inflammation can be recognized. There is extensive loss of tissue through necrosis and suppuration, and healing takes place only after a rather extensive new formation of tissue.

Local infection—Most wound infections are local infections and many specific infections are local or at least localized. There are, however, a number of conditions due to infectious agents to which the term local infection would seem to apply more correctly than any other. A local infection is one in which the organisms remain and exert their action within a limited area. Neither the organisms nor their toxins enter the circulation or cause in any way systemic disturbance. When strictly local infections do cause systemic disturbance, it is usually due to secondary complications or to the toxicity of the toxicoid elements that come from the disintegration of tissue cells and retrograde changes taking place in the area of local infection and finding their way into the system. In those cases that we would term local infection, the mode of entrance is not definitely known. Examples of local infections are such conditions as fistula of the withers, poll evil, necrotic stomatitis and infectious rhinitis in pigs, infectious keratitis and infectious suppurative cellulitis in cattle; thrush in the horse, actinomycosis and many cases of pyogenic infection.

A localized infection is one in which the organisms remain in a given area or structure, and cannot be demonstrated in

the blood, yet there may be marked systemic disturbance (toxemia) due to the formation and liberation of a toxin by the micro-organism constituting the infection. Examples of those conditions that we would term localized infections are tetanus, malignant edema, strangles, chicken pox, ovine caseous lymph adenitis and most cases of pneumonia.

Fever

The pathological process known as fever offers for study and research one of the most interesting and important phases of infection. Fever resulting from infection first manifests itself by a rise in body temperature. Pyrexia is looked upon as being evidence that foreign protein is being broken down although it is generally believed that bacteria possess, or produce, a fever-producing substance, yet all attempts to isolate it have failed. If we reason wholly on the basis of those factors that are normally concerned in heat production, then pyrexia is the result of an abnormal oxidation and tissue disintegration. The old theory was that pyrexia was an indication of, or rather, an attempt on the part of the body to counteract the cause of the fever. This leaves undecided the question as to whether pyrexia in fever is in itself harmful, or beneficial to the infection.

Let us assume that increased body temperature, in part, results from the activity of the infectious agent in its injurious action on the structures of the body, and in part from the activity of the protective forces in their action against the injurious agent and its products. In this light pyrexia in fever may be looked upon largely as a phase of the reactive process the same as exudation in inflammation, and hence is, at least, indirectly harmful to the infection or cause of the fever, and therefore, beneficial to the tissue or body as a whole. That the increased body temperature during the course of an infectious fever is a sort of exhaust or safety valve seems to be borne out by the way pyrexia is dealt with clinically. Beyond a certain maximum temperature life is endangered. On the other hand, the temperature is never through the use of medicines suddenly reduced to normal, but is kept, if possible, within a certain minimum and maximum which suggests that through increased body temperature there comes a certain amount of relief. The question as to whether the pyrexia does, in itself, check or facilitate the action and course of the infection is still undetermined.

The entrance of micro-organisms or their products into the animal body is in those cases constituting an infection followed by activity of the organism, destructive of body elements and stimulating the activity of the protective forces of the body. This combined action results in fever, the charac-

teristic evidence of which is a rise in the body temperature (pyrexia). From the entrance of the infective agent until it is finally destroyed and eliminated from the body there are, in most cases, and particularly in specific infectious fevers rather definite and characteristic changes as a result of this inter-action between the micro-organisms and the animal body.

From the entrance of the infection until disturbance is evident in the tissues, either local or general, there is a period known as the period of incubation during which nothing can be observed. During this period, however, the bacteria have been growing and forming toxins which when formed in sufficient quantities, begin to have an injurious effect upon many of the structures with which they come in contact and to stimulate the tissues to a reaction that is protective in function. These protective forces continue to develop strength, reaching their maximum in the last part of the period of fastigium. No doubt many important changes take place in the body during the period of incubation. They represent the beginning of hostilities between the action of the invading micro-organisms and the protective forces of the body, and may be demonstrated chemically and histologically. The time at which these changes become sufficiently marked to produce clinical symptoms represent the termination of the period of incubation and the beginning of disease or onset of the fever. The onset may be sudden, gradual or very gradual, varying in different diseases in different individuals and in different outbreaks of the same disease. The period known as the onset terminates when the fever (reaction) has reached the high point typical for the particular infection, and beginning here, we have a period known as the stage of fastigium or high ridge. It represents the time of the height of the reaction or the period of maximum activity between the destructive forces of the infection and the protective forces of the body. It is the time when the maximum destruction of cells and tissue occurs. The retrograde change consists largely of a granular degeneration of the parenchymatous structures including, in many instances, the blood. In mild cases, the degeneration of cells is not beyond the point where recovery or repair is possible. In fatal cases the damage is often extensive, and barring those cases where other factors enter in as the cause of death, is beyond repair even had the individual lived. In those cases that come in between the mild and the severe, the degeneration of the cell is less marked, usually not beyond repair, or if beyond repair of the cell itself not beyond the possibility of regeneration of the tissue. The destruction of cells may be confined to areas in the organs and not sufficiently extensive to prevent the part from performing its physiologic function. In this connection it is interesting to recall how often in both men

and animals, although the individual recovers, there is marked clinical evidence of a general weakened condition of some particular organ or part. Beginning with the decline of the fever or the period of defervescence, we are to understand that the various antibodies have been produced in time and quantity to stop the growth, check the action, and in general to establish conditions that are unfavorable to the development and action of the invading micro-organisms and their products. The period of decline may be by crisis or lysis and depends apparently on the quantity and action of the protective elements. In those cases of infectious fevers where the decline is, in the majority of instances, by crisis it can be explained that the cells have been engaged in the production of antibodies which are suddenly liberated and brought to act in great numbers against the infection, while in the case of the decline by lysis the protective forces and antibodies are produced and liberated more gradually. This is further, no doubt, governed by the virulence of the infection and the natural resistance of the individual. It is also quite possible that in certain infections complications due to secondary factors may tend to prevent the fever from taking its normal course, and still further that the micro-organisms may have been carried to parts of the body which are not readily accessible to the bacteriolytic elements from the cells and fluids of the body.

Following the stage of decline, or when the temperature has returned to normal, we recognize a stage or period designated as the period of recovery or convalescence, during which the processes going on in the tissues are reparative and eliminative. The broken down tissue elements and, in fact, all the elements destroyed during the course of the fever or struggle between the invading bacteria and the counter influences of the body must be eliminated. The reparative processes consist mainly of regeneration of those tissues in which regeneration is possible, and by filling in with connective tissue where it is not.

The pathology of fever, eliminating those clinical manifestations such as temperature, increased respiratory and heart action and nervous disturbances may be summed up briefly as follows: Degeneration of the more highly specialized cells of the body especially of the parenchymatous organs, increased number and activity of the phagocytic cells of the body, increased activity of the lymph glands as filters (a function largely developed during the course of the disease), the action of the anti-bacterial chemical substances that are looked upon as belonging to the blood plasma, and the formation of chemical substances by the cells. Those protective substances known as alexins are believed to be normally present in the

blood, while the protective chemical substances formed from the cells, especially leucocytes and looked upon as enzymes, are produced only during the activity of the infection and are no doubt somewhat different for the different infections.

Further, the blood may suffer morphological changes and the plasma and fluids of the body may be chemically changed. The changed composition of the urine and other substances excreted from the body during the activity of certain infections is due partly to interference with normal metabolic processes and to the presence of abnormal elements in the fluids of the body that represent waste products from the interaction between the invading organisms and protective forces. The elimination of toxic substances through an organ (kidney) may result in extensive renal injury to the disintegrated tissue elements and the loss of functional cells, all tending further to change the composition of the excretion. These secondary disturbances often become far-reaching in effect, and may result in structural changes that become more serious than the primary lesions. (Example, vegetative endocarditis, suppurative arthritis in cases of navel ill.)

By secondary infection is meant the presence in the body, local or general, of a micro-organism that has gained entrance to the tissues through the action of a primary infection. The primary infection has so affected the tissues as to facilitate the entrance of the secondary infection or, in other words, has destroyed the natural normal resistance of the body to the invading forces of the micro-organism that constitutes the secondary infection. The primary infection may be local or general, specific or non-specific, and the second infection may be specific or non-specific resulting in local or general lesions, or the primary and secondary infection may be both specific or non-specific. It also happens that in some cases more than one species of bacteria are involved in the secondary infection.

Hog cholera and tuberculosis furnish good illustrations of specific infection where the secondary infections play an important part. In hog cholera pneumonia from a secondary infection is frequently encountered. On the other hand, pneumonia apparently would in no way favor or hinder the entrance of the hog cholera virus.

By mixed infection we mean the entrance into the body of two or more species of bacteria. Both species must be capable of causing infection and shall have entered the tissues simultaneously.

Most wound infections are mixed infections. Of the specific wound infections, tetanus is an instance where pyogenic cocci frequently enter simultaneously with the tetanus baccillus. It is not always possible to differentiate between con-

ditions that result from secondary or from mixed infections, however, where possible it is often a great help in understanding the lesions and in directing treatment. Those pathological processes resulting from secondary, mixed or double infections are always more serious than lesions resulting from a single species of bacteria. This is due to the fact that the pathologic properties of different species are not antagonistic for there is ample evidence to show that the association of two or more species of bacteria tends to increase their virulence, or what is probably more often true, that the combined action of the organisms greatly lowers the resisting powers of the body. Therefore, while the virulence of a particular species may not be increased, the normal barriers are broken down and thus their spread and action are unchecked.

Secondary localization of infectious agents may take place in cases of both specific and non-specific infections. By this is meant an infection that is more or less characteristically confined or active in a given structure, either by its apparent selective action or possibly through the channel by which it gained entrance to the body and has been transferred by the process of metastasis to some distant structure not normally invaded by it and there causes lesions. The lesions are often identical with those occurring in the primary infection, or they may automatically vary considerably due to attenuation of the infective agent but more often varying because of the nature of the tissue in which lodgment took place. Example: Stragles, navel ill, vegetative endocarditis and various non-specific forms of infection.

Many times during the course of a disease complications arise that we are unable to explain. Upon post mortem examination lesions are found, sometimes mild in extent, at other times sufficiently extensive plainly to warrant consideration. In such cases it is always a question as to what extent these isolated lesions were responsible for the symptoms and for the morbid anatomy. Again, in animals that have died from acute diseases there are often found, upon post mortem examination, acute or chronic local lesions that apparently had no direct connection with the etiology of the disease that caused the death of the animal, yet may have had sufficient effect upon the corporeal resistance of the individual as to materially help to bring about a fatal termination in cases that otherwise would have recovered. This is a very important factor in both medical and surgical cases, but before any one would be justified in drawing too definite conclusions he will need to carry on a rather extensive study and gather data from a large number of post mortem examinations. Take for example, the bacillus colicommunis, one of the most common organisms

met with as a secondary invader in both local and systemic disturbances in our domestic animals. From more or less recent study it has been shown to have considerable pathogenic properties. We know that this organism is a normal inhabitant of the intestinal tract, and that it as a rule only invades the tissues by gaining entrance through an injury or from lowered resistance. If upon post mortem examination we find this organism widely distributed in the body, the question arises was death due to a final septicemia from infection shortly before death, or if not, what influence had the organism in bringing about death. At present we can only say that the bacillus often gains entrance to the animal body during the course of certain diseases, and has sufficient harmful influence to bring about a fatal termination in cases that otherwise might have recovered. At other times the colon bacillus is primarily the cause of certain pathological changes, having gained entrance through wounds and natural openings. This brings us to the point which shows the necessity of completely understanding the pathogenic properties of some of the more common micro-organisms. The colon bacillus is injurious to the tissues in a mechanical way; by causing necrosis, pus formation and often marked tissue reaction and fibrosis. The toxin derived from the colon bacillus is described as an endotoxin, and its action is almost wholly local. It varies in strength, a fact which accounts for the variation in the injury and reaction in cases of infection by the colon bacillus, therefore, in infections with the colon bacillus we get more or less necrosis of all structures in the immediate area, the more highly developed or parenchymatous cells being destroyed first and next the fibrous structures. The necrosis is usually not extensive and repair takes place following death of the organisms and neutralization of the toxin. In some cases typical abscesses may be formed. The tissue reaction to the colon organism consists of an exudation of fluid and polymorphonuclear leucocytes. The presence of these two elements together with the occurrence of necrosis explain the tendency to pus formation. Other varieties of white blood corpuscles and fibrin are sometimes more or less abundant. Congestion of the surrounding blood vessels and hemorrhages are not uncommon. There are many cases on record where the colon bacillus has entered the urinary tract, gall bladder, liver and pancreas, resulting in mild chronic inflammatory changes. The lesions resulting from this form of infection are then necrosis and degeneration of the more delicate structures, pus formation and chronic inflammatory processes. They are probably more frequently chronic than otherwise, and in cases where the individual is resistant the only remaining evidence of the lesion produced by the colon bacillus is a sclerosis. I wish to em-

phasize then that upon post mortem examination the finding of sclerotic area in the liver, kidneys, etc., or of old abscesses would suggest that at some previous time infection by the colon bacillus had occurred.

Summary

We are at the present time more or less familiar with the biological and chemical properties peculiar to the various micro-organisms causing infections, the conditions and channels favorable to their entrance into the animal body, the type of injury or general microscopical and gross anatomical changes that result from their activity and finally in most cases the character of the reaction that results from the activity of the various protective mechanisms at the disposal of the body to oppose them. Many of the changes making up the lesion both from the injury and the reaction, especially the reaction, are fundamentally chemical in nature and of them we have very little definite knowledge. Further light on this phase of infection and fever will come from a more complete understanding of the chemical processes in physiology through a correct interpretation of natural biological laws both for micro-organisms and the animal body but largely through chemical research along the lines that have already been pointed out in the science of immunology. In the majority of infections and especially the infectious fevers the most important phase of the reaction is the activity of those factors that have to do with the formation of antibodies and their action against the invading infectious agent and neutralization of toxins.

FURTHER STUDIES OF THE TEETH AND SINUSES

(Second Paper)

(Lantern Slide and Specimen Demonstration)

DR. H. S. MURPHEY, AMES, IOWA

In our descriptive work we use the term median plane, which divides the body longitudinally through the vertebral column. It is at right angles to the ground plane.

Planes on either side of this and parallel to it are called sagittal planes.

Transverse planes divide the body into two unequal parts and are at right angles to the median plane, the head being described in the horizontal position; *but our transverse plane of the head is at right angles to the face.*

Horizontal planes are parallel to the ground plane. We use the term dorsal for superior, ventral for inferior, medial for internal, lateral for external.

Fig. 1 first report is a sagittal section of the head of a five-year-old skull to show the divergence of the teeth. The interalveolar septa are the same except that between the fifth and sixth. Note the infraorbital canal, also the lateral portion of the anterior maxillary sinus above the fourth and fifth cheek teeth. The canal and this bony septum which is quite evident above the fourth separate this portion of the sinus from the medial or turbinal portion.

Fig. 35 first report is a lateral view of an eight-year-old skull (8 year, 2 mo., 6 da.) to show the bony nasolacrimal canal which is slightly convex and extends from the superior part of the infraorbital foramen to the bony medial canthus of the eye. Either of these points can be located accurately only by palpation. We will show later by an X-Ray slide that the medial canthus of the lids is considerably higher than the bony canthus (Fig. 78). From two to eight years of age the second cheek tooth lies below and just in front of a transverse plane through the infraorbital foramen. Note the anterior end of the facial crest. A transverse plane through this point always hits the anterior ridge of the fourth cheek tooth at the level of the functional crown and on account of the obliquity of the tooth the roots are posterior to this plane; then the oral ends of the premolars are anterior to the facial crest in the adult.

Fig. 37 first report shows the same head from above. Note the position of the premolars anterior to the facial crest, the direction of the bony nasolacrimal canal from the orbit to the

plane of the infraorbital foramen. The ducts are convergent to a marked extent. From here forward, they are nearly in a sagittal plane.

Fig. 46 is a superficial dissection of the head from above. Note the angular vein of the eye, which can and should be located in operations here by compression above and in front of the anterior end of the facial crest.

Fig. 47 is a lateral view of a superficial dissection of the head. Note the angular vein and the angular artery of the eye with an accompanying vein passing between the two muscles. These two vessels are parallel, but rarely contiguous, as usually illustrated. There may be a small artery and two veins lateral to the sinuses in the operation field. The transverse facial vessels, which are at first superficial, penetrate the substance of the masseter muscle just below the eye.

Fig. 48 is a deeper dissection from the same head. Most of the masseter muscle is removed here. The superficial muscles are cut and laid back. Note the plexus of veins which lie under the masseter muscle.

You see why it is necessary to raise the malar and angular veins of the eye in front and above the anterior end of the facial crest, also the plexus of buccal veins under the buccinator muscle. Note the infraorbital nerve at its emergence, and its anastomosis with the facial nerve. Note particularly the complexity of the vessels and nerves anterior to the facial crest and lateral to the premolars.

Fig. 49 is a sagittal section of the head. Note that the anterior limit of the frontal sinus and the posterior part of the hard palate are approximately in the same transverse plane. Note the declivity of the oral cavity at the fauces under the soft palate. The superior turbinate bone is composed of a posterior sinus portion, a middle scrolled portion and an anterior cartilaginous portion. The inferior turbinate has the same subdivisions, the sinus portion forms the turbinal (medial) part of the anterior maxillary sinus. The posterior part of the section is to the right of the median plane, so that the right arytenoid cartilage and vocal cord are shown.

No. 6 is the eustachian orifice in the lateral wall of the pharynx.

The gutteral pouch is above, the median septum being cut away anteriorly.

Fig. 50 shows the mucous membrane of the nasal septum, medial view, cartilage removed to show the veins converging postero-medially. This peculiarity of the venous circulation will be seen better in the cross sections. (See Fig. 101 et. seq.)

Inherited defects of the enamel on the developmental and

occlusal grooves may or may not be the primary seat of caries as pointed out by Noyes and others.

Fig. 26 first report is a lateral view of a two-year-old skull. Note the position of the *second tooth in front and below the infraorbital foramen. This is shown to compare with the eight year old (Fig. 35 first report) as a definite land mark between these two ages for the exact location of the second cheek tooth.* See Fig. 28, first report, which is the same head viewed from above to show the sinuses and the location of the teeth. Note that the fifth is just in front of the orbit and that the fourth is under the anterior end of the facial crest.

Fig. 29, first report, is a lateral view of a three-and-one-half-year-old head with all of the teeth erupted. However, the third and sixth may not erupt until four years of age. (Sisson.) Note the thick interalveolar septum between the first and second cheek teeth dorsally, and the exceedingly thin one between the fourth and fifth cheek teeth; (we have found this thin septum in one case between the third and fourth, see Fig. 70). From this age on to five or six years the embedded dorsal portion of the second, third and fourth teeth move forward so that the interalveolar septa at that age are similar as pointed out on the five-year-old head. All of the teeth show developing roots except the sixth. I want to call attention particularly here to the fact that the fourth superior tooth is not as high dorsally as the third and fifth. This fact will be referred to later in the four years and ten months head (Fig. 71), but is present from this age on; it is well marked both above and below at six years. (See Fig. 72.)

See Fig. 31, first report, which is the same head from above. The second tooth is under and anterior to the infraorbital foramen. Note the very thin septum between the fourth and fifth; the sixth is still under the orbit.

Fig. 33 first report is a lateral view of a six year old to show the typical arrangement of the septum between the two maxillary sinuses; also see Fig. 36, first report. Note that ventrally it is transverse in its entirety, while dorsally it is oblique upward and backward superficially, while the plate from here extends backward and inward. We shall demonstrate on the sections of the gross specimens presently the topographic result of that, for example, a transverse plane through here would show the frontal sinus, below the posterior part of the two subdivisions of the anterior maxillary sinus and lateral to these the anterior part of the posterior maxillary sinus. (See Figs. 106, 107 and 108.)

Fig. 39, first report, is a twelve-year-old head to show partial subdivision of the lateral portion of the anterior maxillary sinus. This plate of bone extending above the lateral

roots is persistent. We believe this a plausible explanation of the recorded cases of complete subdivision here. We recorded one case in the proceedings last year. Since that time we have examined a large number of heads and have been unable to find such a blind pouch and doubt its existence. Figs. 53, 95 and 96.

Fig. 30 first report shows an unusual location of the septum behind the fifth cheek tooth. We have no idea how frequently this occurs. Note that in this case the anterior maxillary sinus does not reach the anterior end of the facial crest.

Figs. 52 and 53 show one of the cases in which the septum reaches the anterior end of the facial crest. In these cases the septum is oblique, upward and backward and inward. We observed an unusually high percentage of these in the dissecting room this year.

Fig. 54 is a horizontal section of the head split from the angles of the lips backward. Note the lateral convexity of the cheek teeth and that they are divergent, wider behind than in front.

Fig. 55 is the lower part of the same section, showing that the mandibular teeth of a particular side are approximately in a straight line and that the two arcades diverge as they go backward wider behind than in front, with no appreciable lateral curvature.

Figs. 56 to 77 are made directly from radiograph negatives except Figs. 63, 65 and 68, which are from photographs, while Figs. 78 and 79 are made from radiograph prints of sagittal sections of heads of known age. We have radiographed a 101-day fetus (forty millimeters from anterior margin of orbit to tip of nose and twenty-six millimeters transverse diameter at the level of the facial crest). No evidence of tooth germs and very little ossification in maxilla and mandible is present.

Fig. 56 is an 163-day fetus. Note the tooth germs of the deciduous premolars at this age. Calcification is just beginning on the folds forming the cusps.

Fig. 57 is an 179-day fetus. Calcification is advanced on the temporary premolars and is apparent on the central incisors (pincers). These two slides show the bony cochlea and semicircular canals of the petrous temporal bone.

Fig. 58 is a 235-day fetus. Calcification previously noted is considerably advanced. In addition, the intermediate is calcifying. Note that the pincers are very wide antero-posteriorly.

Fig. 59 is a 251-day fetus. The principal change noted here is the rotation of the "pincers" on a vertical axis and the further calcification of the intermediate incisors.

Fig. 60 is the right half of the head of a 280-day fetus, teeth not erupted, two temporary incisor, three temporary premolar germs present.

Fig. 61 is the left half of 308-day fetus head, the maxillary and spheno-palatine sinuses containing a few shot. The anterior maxillary contains the larger shot, teeth erupted. Note the plicated pattern and similarity of the superior and inferior cheek teeth.

Fig. 62 is the right half of head of a 340-day fetus (end of term). The third temporary incisor tooth germ is present. The first molar germ is hazel-nut size, but is not calcified enough to show in the radiograph.

Fig. 63 is the lateral view (from photograph) of same head with soft structures laid back and sculptured to show teeth. The premolars do not show the detail here that is shown in the radiograph, but note the first molar germ which does not show in the radiograph.

Fig. 64 is the right half of an 107-day-old colt, the third incisor calcified, but not erupted. All temporary premolars are erupted, first molar 3.5 cm. long, second molar germ 1 cm. in diameter above, below it is still a tooth band. The boundaries of the sinuses are plainly shown by the light areas. The anterior limit of the sinus here is over the middle of the third tooth, while in the 280-day fetus previously shown it was over the middle of the second. While the sinus itself is enlarging (moving forward in terms of the surface landmarks), the teeth are moving forward under it at a greater rate of speed. (The gross specimens of the other half of this head were exhibited in cross sections to show the exact pattern of the sinuses.)

Fig. 65 is a photograph of the same head with soft structures laid back and sculptured to show teeth. Note germs of second molar, also the infraorbital foramen dorsal to the anterior root of second temporary premolar. The trephine openings above the teeth were used to fill the sinuses with shot for radiographing.

Fig. 66 is the right half of seven-month-old head. Incisors are all erupted, corners just coming into wear. No evidence of the permanent premolars found. First molar above 7 cm. long, second 3 cm. long. The corresponding teeth below are a little shorter. The third molar ridge below is 0.5 cm. deep, above it was not found; first premolar (wolf tooth) present above. Note that the infraorbital foramen is now above middle of second temporary cheek tooth and that the dorsal part of the first molar is just anterior to the orbit.

Fig. 67 is the left half of an eight months and seventeen days old colt. $PM\frac{1}{1}$, the "wolf tooth," is 1 cm. anterior to the second premolar (first cheek tooth); measured on the superior border of the mandible, PM– (wolf tooth) is also 1 cm. anterior to $PM\frac{-}{2}$ (first mandibular tooth). Note the abnormal reserve crown (beginning odontoma?) of the first molar; also that the first molar both above and below has already erupted through the gums.

Fig. 68 is a seventeen-month-old head (small horse—"scrub") sculptured (the fifteen-month head, Fig. 23, first report, is probably nineteen months old). Note that the fourth premolar is halfway under the anterior end of facial crest and that the second molar is just anterior to margin of orbit. The first and second permanent cheek teeth above, and the first below, were about one inch long; no evidence of the second below, first molar above 9 cm. long, second 6.5, the third not found.

Fig. 69 is the left half of twenty-three-month-old head; animal underdeveloped; tooth germs of permanent premolars and third molar comparatively small. *This may explain variation in the time of eruption. However, until more data is collected, we shall believe that there is a mean rate of growth for each tooth and that the time of eruption depends primarily on the time of germ formation more than on the state of nutrition. We have previously noted that the fourth premolar germ below is the last to form, but usually erupts before the corresponding one above, or the third molar either below or above.* The extent of the sinuses is quite evident here as well as the mandibular canal. Note $PM\frac{1}{1}$ (wolf tooth above and below).

Fig. 70 is a two-year, seven-month, thirteen-day-old head. Note that the temporary premolars are still present, although the anterior part of the second above is broken off. (See Figs. 99 to 110 for a cross section at this age; note the $PM\frac{1}{1}$ [wolf tooth both above and below]).

Fig. 71 is the right side of a four-year, ten-month-old head. The interalveolar septa are similar except the oral half of that between fourth and fifth superior cheek teeth. The roots of the mandibular teeth show a greater divergence than the maxillary teeth. *Note contact of superior and inferior teeth. The sixth tooth below is wider than the opposing above, hence it comes in contact with the sixth and fifth above, the fifth below with the fifth and fourth above, the fourth below with the fourth and third above, the third below with the third and second above, the second below may or may not come in contact with the first above, always with the second above. (See Fig. 51.) Note also that the fourth cheek tooth is shorter than either the third or fifth.*

The roots of the superior one, though longer than its contiguous teeth, are still a little more than one centimeter lower than the third and fifth. All of the teeth show developing roots except the sixth. The margin of the orbit and the first portion of the bony naso-lacrimal canal are quite evident. Note extent of sinuses, the anterior maxillary reaching the middle of the third cheek tooth.

Fig. 72 is a five-year, eight-month, eleven-day head; roots well marked on the sixth below. Note that the fourth is considerably shorter than the adjacent teeth; also the forward movement of the imbedded parts of the second, third and fourth. We now believe that the explanation of this phenomenon is probably the "lift" on the posterior part of the tooth, during mastication, by the anterior part of the next one of the mandibular series, thus pushing the fang end forward; e. g., the fourth above is "rocked" forward by the fifth below (see page 153, A. & B., first report). We also believe that the frequent breaking of the fourth above is due to: first, incomplete cementation with resulting decalcification due to acid-producing bacteria which live here in the recesses on decomposing food particles; second, the uneven stress it is given by the (small) fourth below and the (strong) fifth below (see Fig. 51); and third, the antero-posterior "squeeze" exerted at the functional crown due to the obliquity of the series, which is constantly present as a growth factor, but is increased many times during actual mastication, fourth incomplete union of the dentinal plates (which we have observed more times in this tooth than in all others combined) probably due to disturbed vascular supply (nutrition). (See Figs. 12 to 16, inclusive, and page 147, first report.) The breaking or splitting will be in a transverse plane (to the median plane of body) or obliquely taking off one corner when only one infundibulum is incompletely cemented (as in fifth tooth, Fig. 16, first report), or when only one infundibulum is incompletely cemented and complicated by incomplete union of the dentinal plates of the antero-medial column of the tooth (as seen in the fourth tooth of the right arcade, Fig. 14, first report). The splitting will be in a sagittal plane when both infundibuli are incompletely cemented.

Fig. 73 is a seven year old (exact days unknown). The fang development is quite evident on the mandibular teeth. The teeth are comparatively small for a pure-bred draft horse.

Fig. 74 is a Hackney stallion ten years and four months old. Notice that the fangs are becoming pointed and that the apical foramen is about the diameter of the lead of a pencil. (Ca. 1mm.)

Fig. 75 is a twelve-year-old mule. The effect of "champ-

ing the bit" is well shown on the first cheek and mandibular teeth. The remainder of the teeth are comparatively longer than in a horse of the same age. The second and third cheek teeth above have three fangs.

Fig. 76 is a fourteen-year-old head. Note the hyoid bone and its position during deglutition.

Fig. 77 is a sixteen-year-old head. Note the root development, particularly of the mandibular teeth, orbital margin, first part of bony naso-lacrimal canal, the infraorbital canal and the recession of the teeth from it. Note the great cornu and thyroid cornu of the hyoid bone. The horse died in deglutition, hence they are forward.

Fix. 78 is the head of an old horse. (Note the position of the hyoid bone in a state of rest.) The naso-lacrimal duct was injected with opaque material. Note its sinuous outline and its dorsal curvature behind the infraorbital foramen. Both lacrimal ducts are injected and *this slide shows that the inner bony canthus is ventral and slightly anterior to that of the lids; as previously noted the bony canthus is the reliable landmark and should be located by palpation.* If the trephining should be done with the palpebral canthus as the landmark one would be likely to injure the naso-lacrimal duct. The infraorbital canal and the parotid duct contain wires. The artery is only partly filled, but note that it is in front of the duct.

Fig. 79 is a radiograph of a vascular injection.

A. The common carotid artery.
   I. The occipital artery which anastomosis with the (v) vertebral on the dorsal aspect of the wing of the atlas.
   II. The internal carotid artery.
   III. The external carotid artery.
      III. A. External maxillary artery.
         1. Pharyngeal artery.
         2. Lingual artery.
         3. Sublingual artery.
         4. External maxillary winding around border of mandible to become the facial artery.
         5. "Anterior masseteric artery."
         6. Inferior labial artery. Six' prime superior labial artery. (Other branches not shown. See Fig. 47.)
      III. B. Masseteric artery ("posterior masseteric artery.")
      III. C. Posterior auricular artery.
      III. D. The superficial temporal artery.
         7. Anterior auricular artery.

8. Transverse facial artery.

III. E. Internal maxillary artery.

9. Inferior alveolar artery.
10. Pterygoid arteries.
11. Posterior deep temporal artery.
12. Anterior deep temporal artery.
13. Buccinator artery.
14. Infraorbital artery.
15. The greater palatine artery.

At this point slides illustrating the development of the tooth germ in the embryo were shown, slides taken from Noyes. At about two months of age the space of the oral cavity is present and the dental ridge begins to evaginate from the epithelium lining the oral cavity. A later stage shows beginning of the development of the enamel organ. Invagination begins from below. Further development of the enamel organ. Note that it is still connected here with the epithelium of the mouth. A still later stage, tooth germ in complete outline from the mandible of a sheep embyro.

This is the outer tunic and this the inner tunic, which gives rise to the ameloblasts or adamantoblasts. This is the dental papilla, the outer covering being a row of differentiated cells, the odontoblasts, which will give rise to the dentine which will be deposited centripetally. This represents, then, the pulp cavity containing pulp which will be obliterated by the dentine.

This is a still later stage showing the bud which will develop into the permanent tooth germ. Note that it is connected with the temporary tooth germ and that both are still connected by a band with the epithelium of the mouth. A portion of calcified material is present at the cusps.

This is a tooth germ from an embryo pig which may be used to illustrate the development of the inner enamel ring by plication. It is at the folds that the enamel defects previously shown are likely to occur.

Fig. 80 was made from a microphotograph of a cheek tooth of the horse.

Fig. 81 is a greater magnification from the top of the same section to show developing dentine and enamel, the pulp cavities, infundibulum, layer of ameloblasts which have to do with the deposit of the enamel, also the row of odontoblasts which, as previously stated, have to do with the deposit of the dentine.

Fig. 82 is a small section from the previous slide to show more detail by greater magnification. The dentine is made of parallel rod-like structures. The enamel is also rod-like, the

rods being parallel or gnarled as previously shown. The dento-enamel junction is not a straight line, but uneven. The spaces are artefacts. The uneven line probably gives greater strength to the union of these two structures.

The peridental membrane is divided into three parts: First, the gingival; second, the alveolar portion; third, the apical portion. We used to consider the peridental membrane as a modified periosteum. The gingival portion was considered a part of the submucosa. It has three functions:

First, it attaches the tooth to the surrounding soft and hard structures.

Second, on the alveolar side, it is covered by osteoblasts which have a bone-building function, while on the dental or tooth portion it has a layer of cementoblasts, which build cementum.

Third, the sensation of touch for the tooth is entirely in this membrane.

In addition to the cellular elements mentioned it is made of white fibrous connective tissue (collagenous) fibers and fibroblasts. Osteoclasts are also present, during growth periods, and in certain pathological conditions. It is, of course, supplied with both vessels and nerves. Formerly we considered cementum as modified bone, now we consider it a distinct tissue. It does not have quite the same chemical composition as bone and a much different arrangement (Morphology). Fibers of the peridental membrane pass into and form part of the cementum while their attachment to the bone of the alveolus is superficial and by apposition.

I want to call your attention particularly to the small space between the gum and the tooth which is increased in size by inflammatory and edematous conditions of the connective tissue. In pyorrhea the separation goes down farther between the cementum and the peridental membrane so that the separation of the gum from the tooth is of considerable diagnostic importance. The alveolar attachment of the peridental membrane to the bone is uneven due to the pitted condition of the bone in the young animal. In the older animal the peridental membrane is much thinner due to a deposit of cementum on one side and bone on the other.

(The session removed to the anatomical lecture room for the demonstration of prepared specimens.)

We gave some attention to the number of teeth last year, and also to their irregularities. (See first report.) Figure 84 shows a peculiar placing of the canine next to the incisors, directly against them, with a very thin interalveolar septum.

In some heads the wolf tooth is in front of its usual posi-

tion, against the first cheek tooth. It may be half way between the canine and the first cheek tooth. Fig. 67 shows it 1 cm. anterior to first cheek tooth. Other cases of these irregularities have been recorded also.

Fig. 83 shows congenital absence of mandibular incisors on right side.

Fig. 85 and Figs. 4 and 5, first report, show a seventh cheek tooth above apparently unilateral in each case. It seems rather peculiar that there was no marked infection resulting from the one shown in Fig. 4. When we first had the specimen, both the sixth and seventh were loose, and could be moved in the alveoli, and there was considerable space between the fifth and sixth teeth above. Several pratitioners have told me of cases that they had, one peculiar one in which there was a superior supernumerary in front of the first mandibular tooth, which had grown down until it was injuring the mandible, others in which the supernumerary tooth was in the hard palate medial to the normal arcade.

Fig. 88 shows a retained deciduous incisor between the left corner and intermediate. Dr. Bemis reports this condition and supernumerary incisors (see Figs. 86 and 87) as being comparatively common in the clinic. This left, third mandibular tooth, is malformed and too small. Food had collected here resulting in pyorrhea, since the teeth were not in contact (antero-posteriorly) and movement during mastication was possible. Note also that the antero-posterior line is not a straight line (see Fig. 55 also).

Fig. 89 shows a decrease in the number of incisors to five, also a separation on the right between first and second mandibular teeth.

Fig. 90 is another specimen with only five incisors. It is evident that the right central is absent. I am indebted to Dr. Gubser, of Earlham, Iowa, for the loan of this specimen.

Fig. 91 shows the maxillary alveoli. Note particularly that the lateral two roots are always separate and distinct, and that the medial two roots are usually fused.

Fig. 51 shows the contact area of the teeth. The pivot or fixed point in lateral movement during mastication is at the point of contact of the post-glenoid process of the squamous temporal and the condyle of the mandible on the masticating side, while on the opposite side the condyle of the mandible is drawn forward during the first phase and then backward as the lower jaw approximates the upper.

Fig. 92 is a sectioned head assembled to show the level at which the sections were taken, they are numbered from above

downward 1, 2, 3 and 4. (For a dorsal view of this head see Fig. 17, first report.)

Fig. 93 is a ventral view of section 1. A portion of the cranium is cut off showing a part of the cerebrum, on either side the temporalis muscle. The nasal septum is hypertrophied and sinuous.

Fig. 94 is a dorsal view of section 2. On the right side we have cut below the supraorbital process, showing the bulbus oculi. Note the fronto-maxillary opening, medial to the inner canthus and in the same horizontal plane.

Fig. 95 is a ventral view of section 2. Note that the posterior part of the anterior maxillary sinus (2) is medial to the posterior compartment of the maxillary sinus (1). The sagittal plate of bone is directly over the lateral fangs of the teeth (see page 63). Note the position of the sphenopalatine sinus. When the sphenoidal and palatine portions of this sinus are directly continuous, they open into the posterior compartment of the maxillary sinus over the infraorbital canal. If separated, the palatine still opens here while the sphenoidal portion empties into the nasal cavity through the ethmoidal meati.

Fig. 96 is a dorsal view of section 3. Note the floor of the sphenopalatine sinus, also both compartments of the maxillary sinus, especially the plates (17) above the fangs of the teeth described on the other specimen (Fig. 95) as seen in an old horse. The anterior maxillary sinus (lateral portion, left side) is practically obliterated by an hypertrophy of the lining mucous membrane.

Fig. 97 is a ventral view of the same section. Note particularly the size, shape, extent and relations of the pharynx and guttural pouches.

Fig. 98 is the dorsal view of section 4. Note the floor of the guttural pouch.

Fig. 99 is the lateral view of a two years and eight months old head cut in cross-sections, then assembled. The sections are numbered from before backward 1, 2, 3, 4 and 5.

Fig. 100 is the same head from above. We have not made the sections directly transverse so that the right and left sides of a given specimen will not show exactly the same structures.

Fig. 101 is the posterior view of section 1. Note the anterior scrolled portion of the turbinates. The septal cartilage at this level is wide above, narrows, widens again, and is semicylindrical at the floor. Note the venous plexus on the turbinates, also on the septum, just below its middle. On the right side this is the posterior part of the first temporary cheek tooth, below the section is through the anterior part of both temporary and permanent mandibular teeth.

Fig. 102 is the anterior view of section 2. Note the outline of the section here in front of the facial crest.

Fig. 103 is the posterior view of section 2. Note the outline through the anterior end of the facial crest, and the infraorbital nerve and vessels.

Fig. 104 is the anterior view of section 3. Note the outline here again in front and how it widens posteriorly above the facial crest.

Fig. 105 is the posterior view of section 3. The septal cartilage is cut just behind its posterior attachment to the hard palate. Note the difference in position of the infraorbital nerve in relation to the teeth on the right and left sides, also the small amount of space in the oral cavity. The arrow is in the nasomaxillary fissure between the posterior compartment of the maxillary sinus and the middle meatus nasi. Fig. 106 is the anterior view of section 4. Again note the outline in front of and at the orbit.

Fig. 107 is the posterior view of section 4. Note the oblique section of the eye in this and the following figure (108).

Fig. 108 is the anterior view of section 5. Note the posterior part of the frontal sinus, the posterior part of the nasal cavity, and the cross-section of the lateral masses (cells) of the ethmoid bone. Compare the eustachian orifices with Fig. 49.

Fig. 109 is the posterior view of section 5.

Fig. 110 is a cross-section from the head of an old horse. It is the section between the sections pictured last year as figures 108 and 107, first report. Compare the extent of the sinuses in the old and young head.

All of the figures used in publication are original. A few of the lantern slides were made from existing cuts in the literature. Many original lantern slides and specimens used in the lecture are not illustrated in this publication.

It is a pleasure to acknowledge the help and encouragement given this work by Dean Stange and during his illness, to Vice Dean Bemis, acting head of the Division, to innumerable persons for their kindness in furnishing us data regarding the age, breeding, etc., of the material used in this study; to all past and present student assistants, particularly G. W. McNutt and Max McLeod, for preparation of specimens and some negatives; to the photo section of the college we are indebted for part of the photos and all of the lantern slides, and the prints used in publication.

To my colleague in Anatomy, Dr. J. D. Grossman, I am indebted for aid and helpful suggestions; to him and Max

McLeod belongs the major credit for the radiographs, all of which they made.

We are particularly indebted to Messrs. Wurzer and Edison, of the Homestead Company, of Des Moines, for their excellent work in making the half-tone blocks from the prints and X-Ray negatives.

We are taking this opportunity to ask the profession of this state to aid our further studies by furnishing us small equine and bovine foeti from cases of abortion or accident. They should be immersed in 10 per cent formaldehyde (U. S. P.) and shipped in fluid if under 3 mo. foeti. *Exact age in days is very important.*

Send us the history:

1. *Breeding, weight, age, condition, previous sickness, etc., of both sire and dam.*
2. Cause of abortion.
3. *Date (or dates) of breeding.*
4. *Date of abortion or death.*

Foeti two inches or less should be fixed in the following:

Potassium dichromate	2.50
Sodium sulphate	1.00
Mercuric chloride	5.00
.85% NaCl in soft water....	100.00
Formaldehyde U. S. P.....	5.00

Mix just before using—immerse fetus and *send to the laboratory AT ONCE.*

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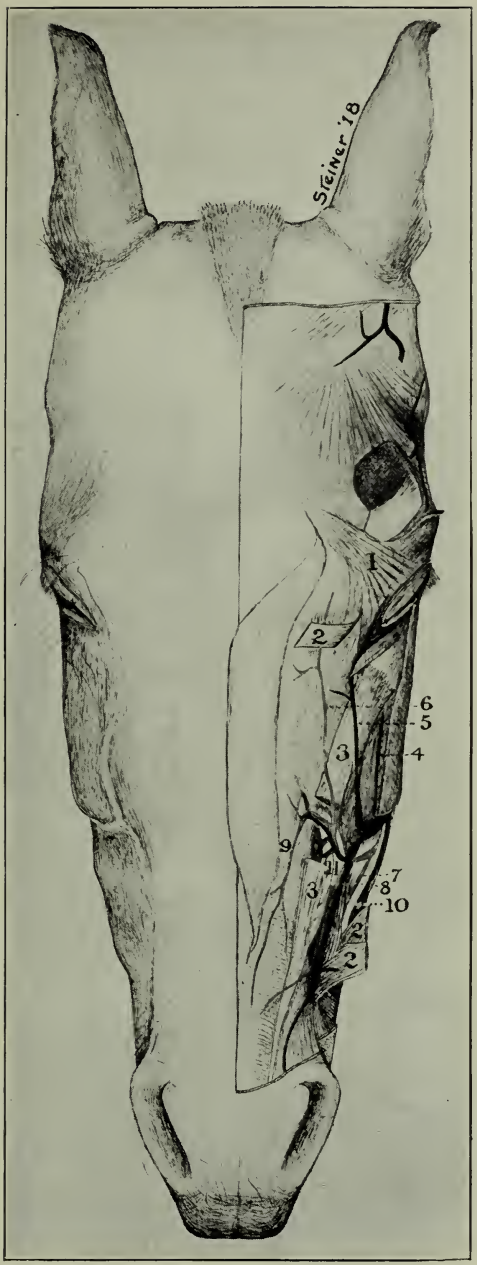

Fig. 46—Superficial dissection of the head from above. The levator naso-labialis is cut and rolled back, sections of the malaris and the levator labii superioris proprius have been removed. 1. M. Corrugator supercilli. 2. M. Levator naso-labialis. 3. M. Levator Labii superioris proprius. 4. Malar vein, varies in size, but constantly present. 5. Angular vein of eye. 6. Angular artery of eye. 7. Superior Labial artery. 8. Superior nasal artery and vein. 9. Dorsal nasal artery. 10. Superior buccal branch of the facial nerve.

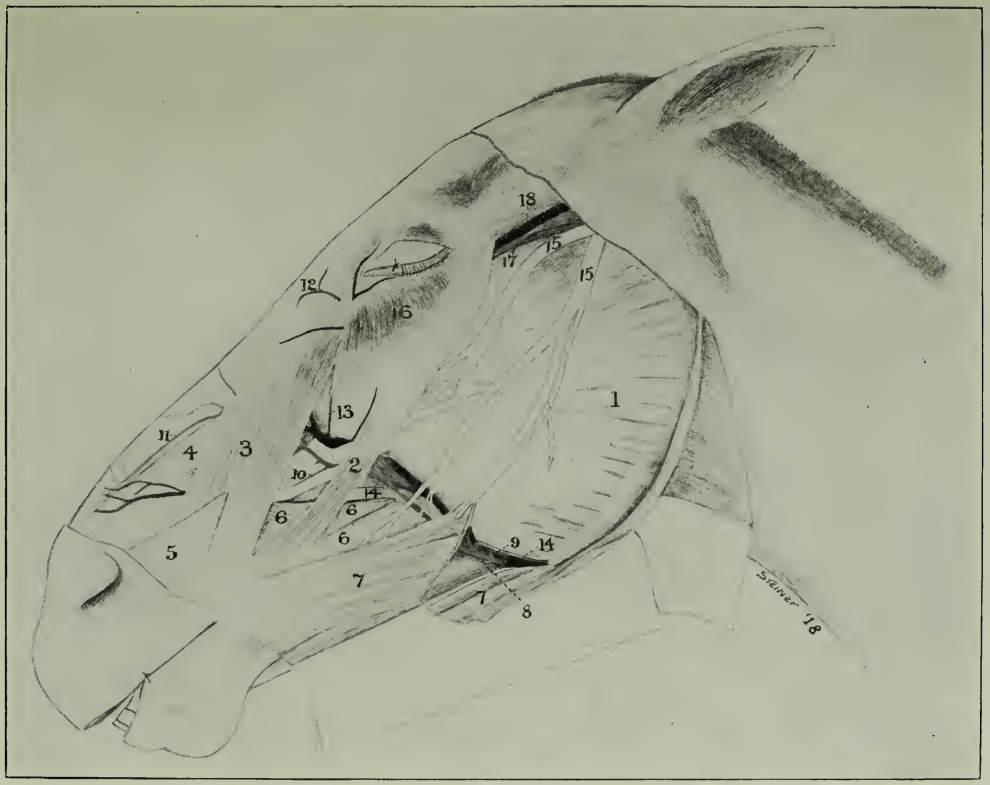

Fig. 47—Superficial dissection of the head, lateral view. 1. M. Masseter. 2. M. Zygomaticus. 3. M. Levator Naso-labialis. 4. M. Levator labii superioris proprius. 5. M. Dilator naris lateralis. 6. M. Buccinator. 7. M. Cutaneous facei (cut to show vessels). 8. Facial artery. 9. "Anterior masseteric artery." 10 Superior buccal artery. 11. Dorsal nasal artery. 12. Angular artery of eye. 13. "Malar vein." 14. Parotid duct. 15. Facial nerve. 16. M. Malaris. 17. Transverse facial artery. 18. Transverse facial vein.

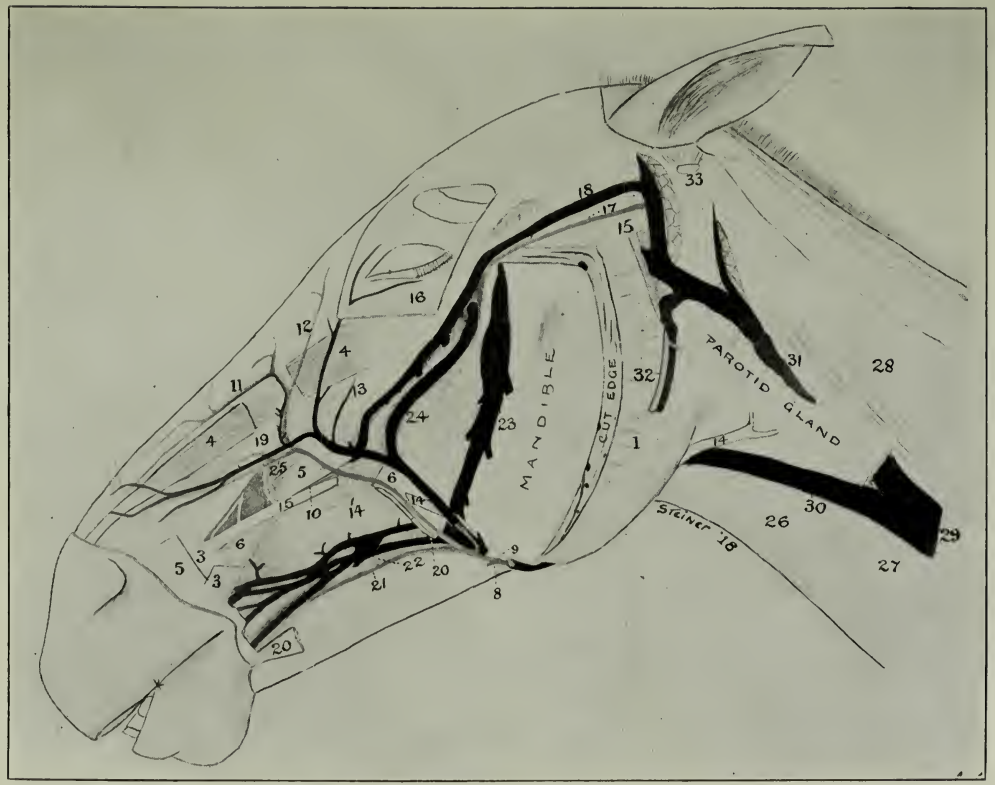

Fig. 48—Deep dissection of head, lateral view. 1. M. Masseter. 3. M. Levator naso-labialis. 4. M. Levator labii superioris proprius. 5. M. Dilator naris lateralis. 6. M. Buccinator. 8. Facial artery. 9. "Anterior masseteric artery." 10. Superior buccar artery. 11. Dorsal nasal artery. 12. Angular artery of eye. 13. "Malar vein." 14. Parotid duct. 15. Facial nerve. 16. M. Malaris. 17. Transverse facial artery. 18. Transverse facial vein. 19. Infraorbital nerve. 20. M. Depressor labii inferioris. 21. Inferior buccal artery. 22. Plexus of buccal veins. 23. Buccinator vein. 24. Vena reflexa. 25. Lateral nasal artery. 26. M. Omohyoidens. 27. M. Sternomandibularis. 28. M. Brachiocephalicus. 29 and 31. Jugular vein. 30. External maxillary vein. 32. Masseteric artery and vein ("posterior masseteric artery"). 33. M. Parotido auricularis.

Fig. 49—Sagittal section of the head. 1. Superior turbinate bone: scrolled portion. 2. Inferior turbinate bone: scrolled portion. 3. Turbinal portion of frontal sinus. 4. Turbinal portion of anterior maxillary sinus. 5. Epiglottic cartilage. 6. Eustachian orifice in lateral wall of naso-pharynx. 7. Soft palate. 8. Frontal sinus. 9. Lateral ventricle of larynx.

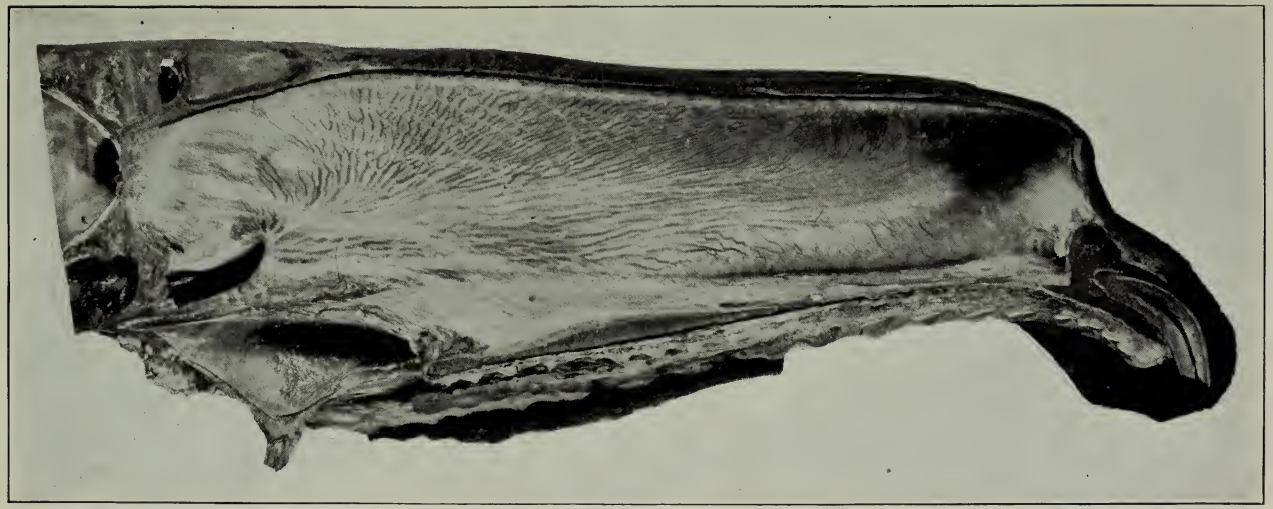

Fig. 50—Veins of the nasal septum, medial view of mucous membrane after removal of septal cartilage (chondral surface.

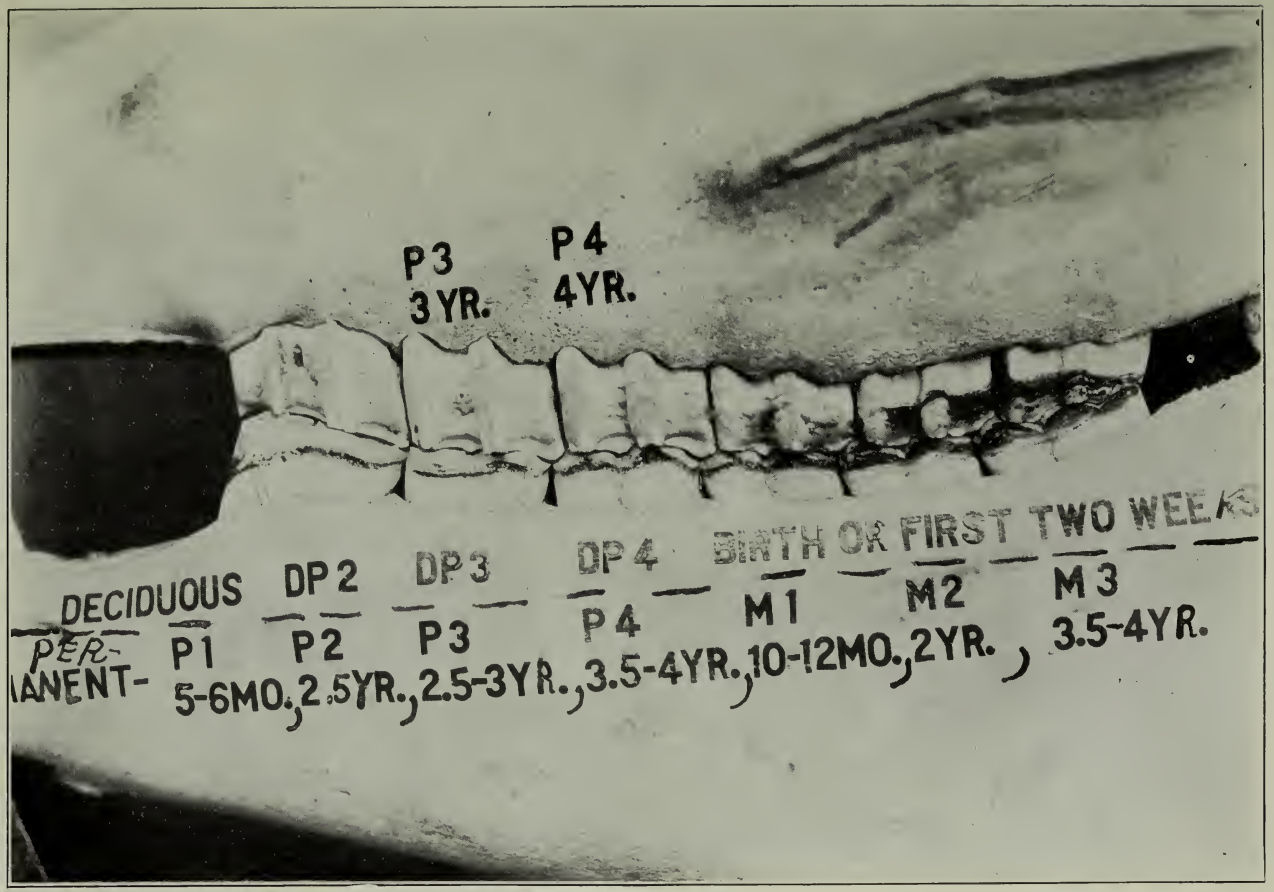
P3
3YR.
P4
4YR.
DECIDUOUS DP2 DP3 DP4 BIRTH OR FIRST TWO WEEKS
PER- P1 P2 P3 P4 M1 M2 M3
MANENT- 5-6MO., 2.5YR., 2.5-3YR., 3.5-4YR., 10-12MO., 2YR., 3.5-4YR.

Fig. 51—Lateral view of skull, mandible pushed laterally to show contact area of the individual teeth of the upper and lower arcades during the last phase of mastication; time of eruption (after table from Sisson).

Fig. 52—Lateral view of head to show septum between the two compartments of the maxillary sinus, extending upward and backward from the anterior end of the faci al crest. 1. Frontal sinus. 2. Posterior maxillary sinus. 3. Anterior maxillary sinus. 4. Infraorbital canal. 5. Bon y naso lacrimal canal. 6. Septum between the maxillary sinuses.

Fig. 53—Medial sections of head, dorsal view. The one on the right is 52 from above. The one on the left shows partial subdivision of lateral (or maxillary) part of anterior maxillary sinus (maxillo turbinate sinus), by a plate of bone dorsal to lateral roots of the teeth. 1. Frontal sinus. 2. Posterior maxillary sinus. 3. Anterior maxillary sinus (lateral or maxillary portion). 4. Septum between the maxillary sinuses.

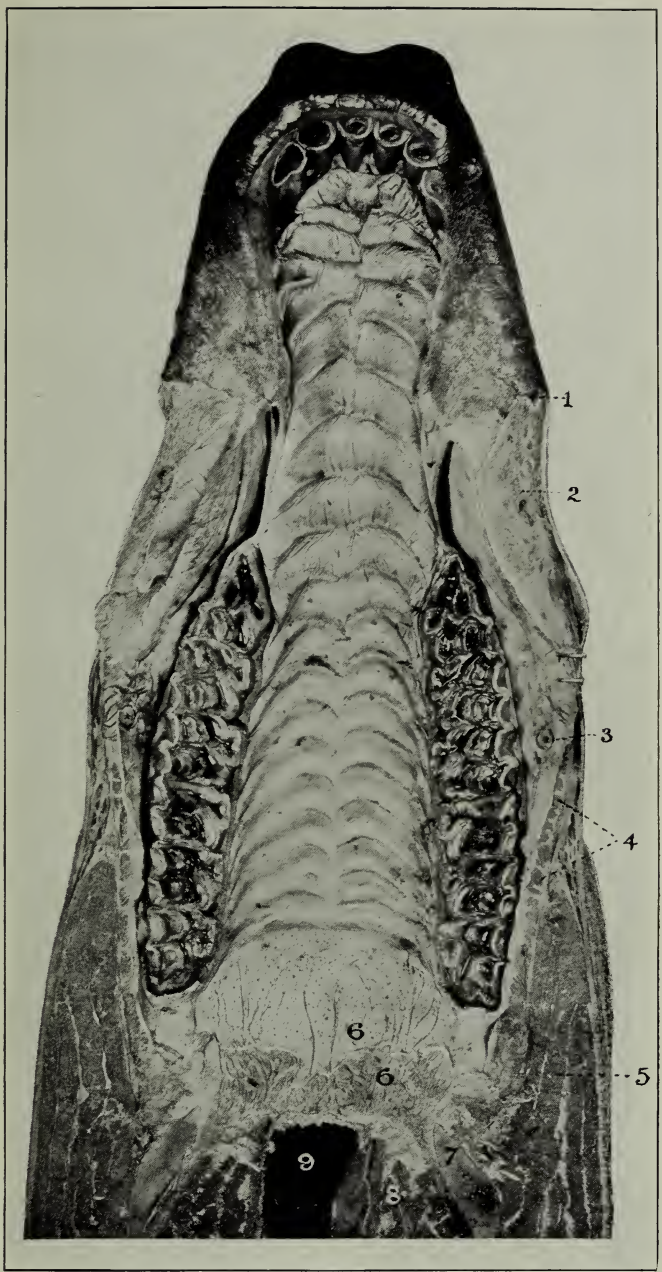

Fig. 54—Horizontal section of heal oral view of roof of mouth. 1. Commissure of the lips (cut). 2. Labial glands. 3. Oral opening of the parotid duct on papilla salvalis. 4. Buccal glands. 5. M. Masseter. 6. Soft palate. 7. Mandible. 8. M. Pterygoideus internus. 9. Pharynx.

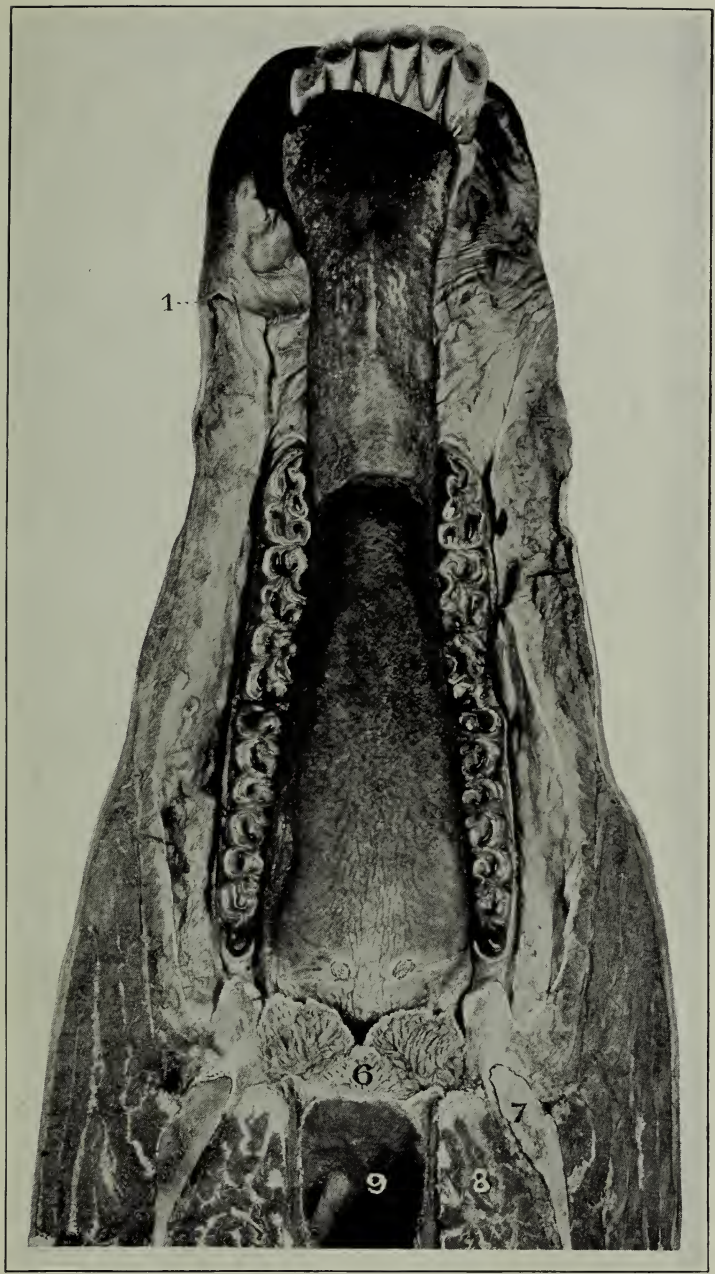

Fig. 55—Horizontal section of head thru rima oris showing the floor of mouth. (For legend see Fig. 54.)

Fig. 56—Radiograph. 163-day fetus. Calcification just beginning on folds of temporary premolars only.

Fig. 57—Radiograph. 179-day fetus. Calcification noted on Fig. 56 is advanced, and is present on central incisors also. Note the developing bony cochlea and semi-circular canals of the petrous temporal bone.

Fig. 58—Radiograph. 235-day fetus. Calcification noted in Fig. 57 advanced and is well marked on intermediate incisors.

Fig. 59—Radiograph. 251-day fetus. Calcification progressing. Note the small "interdental" space, especially in mandible.

Fig. 60—Radiograph of a 280-day fetus.

Fig. 61—Radiograph of a 308-day fetus. The larger shot are in the anterior maxillary sinus, the smaller in the other sinuses.

Fig. 62—Radiograph of a 340-day fetus (end of term).

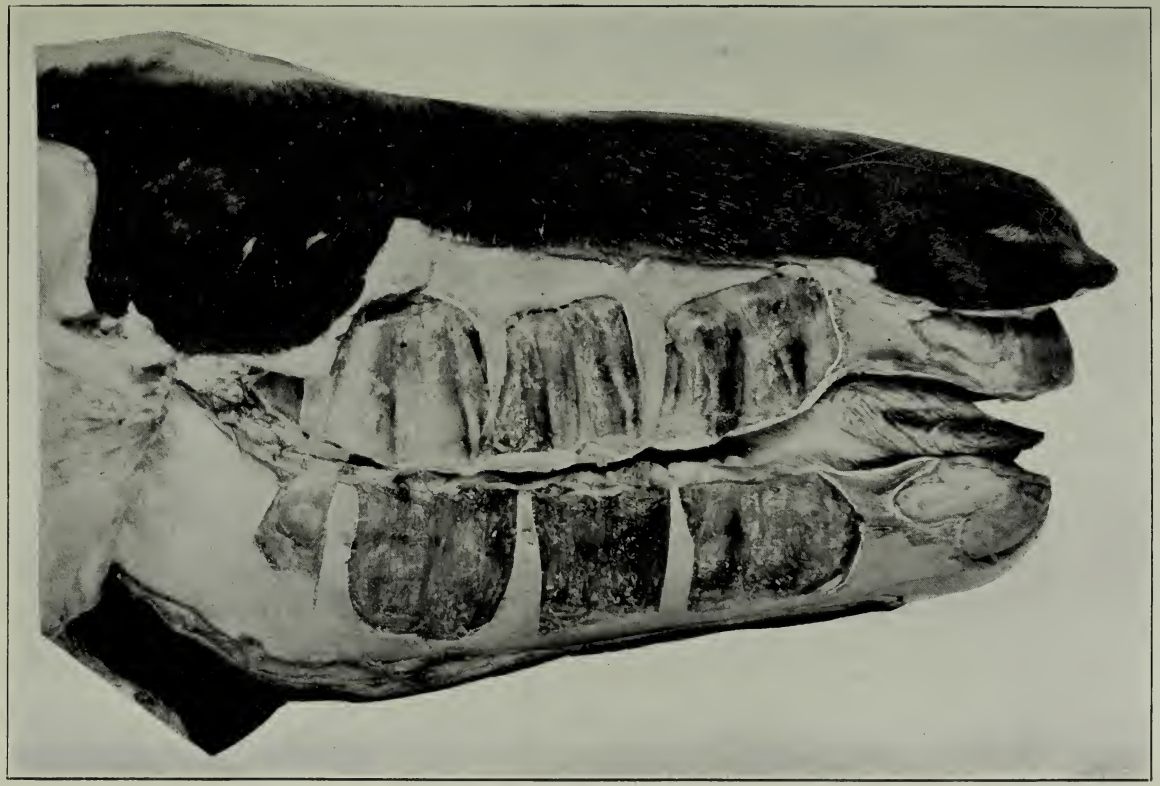

Fig. 63—Sculptured head of 340-day fetus.

Fig. 64—Radiograph of a 107-day colt.

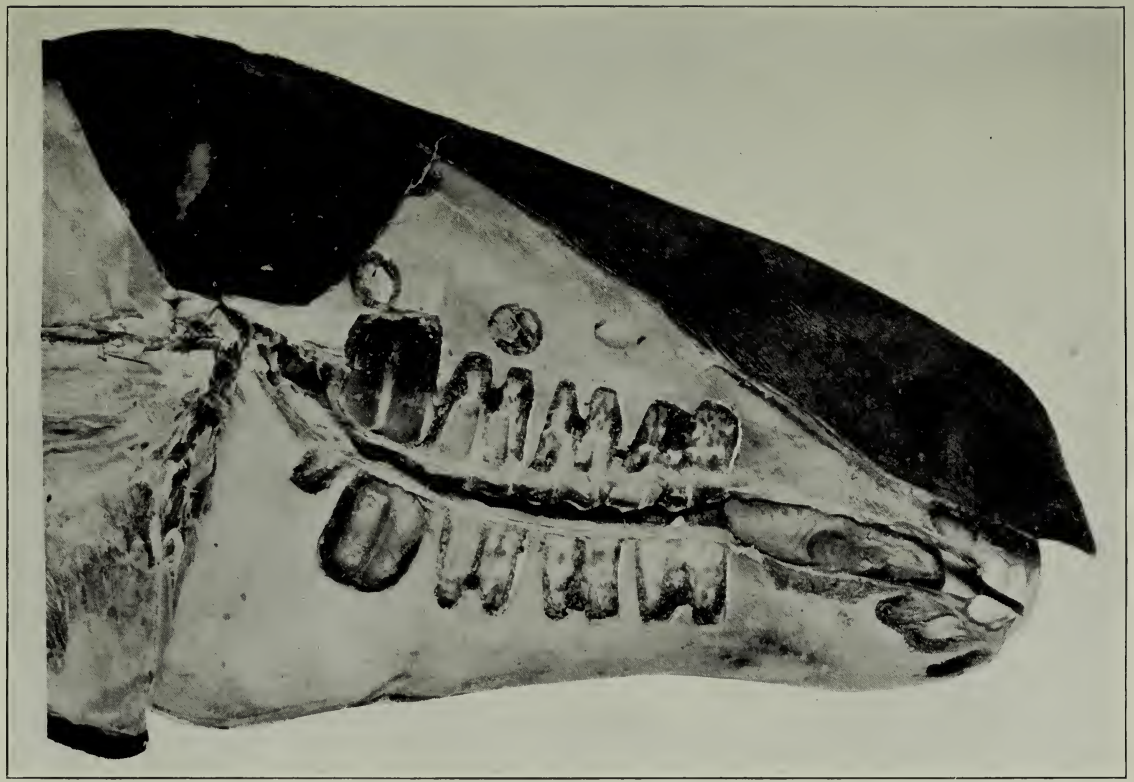

Fig. 65—Sculptured head of a 107-day colt.

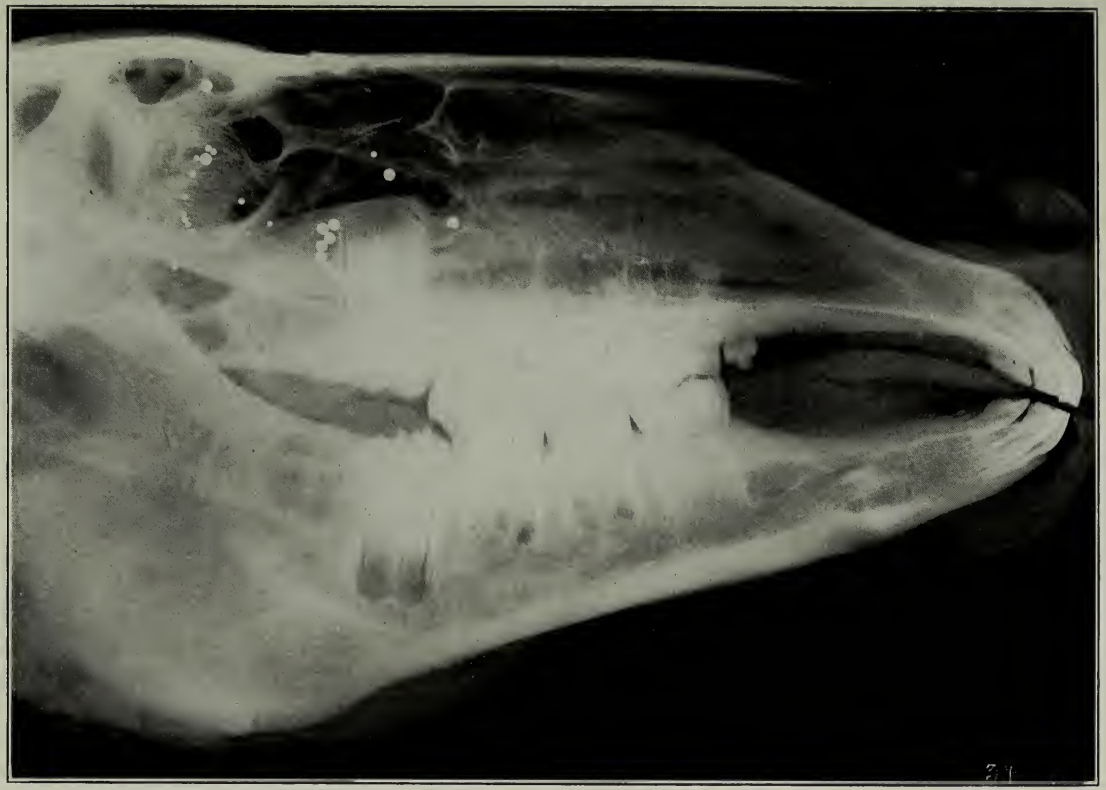

Fig. 66—Radiograph of a seven-months colt.

Fig. 67—Radiograph—eight-month 17-day colt.

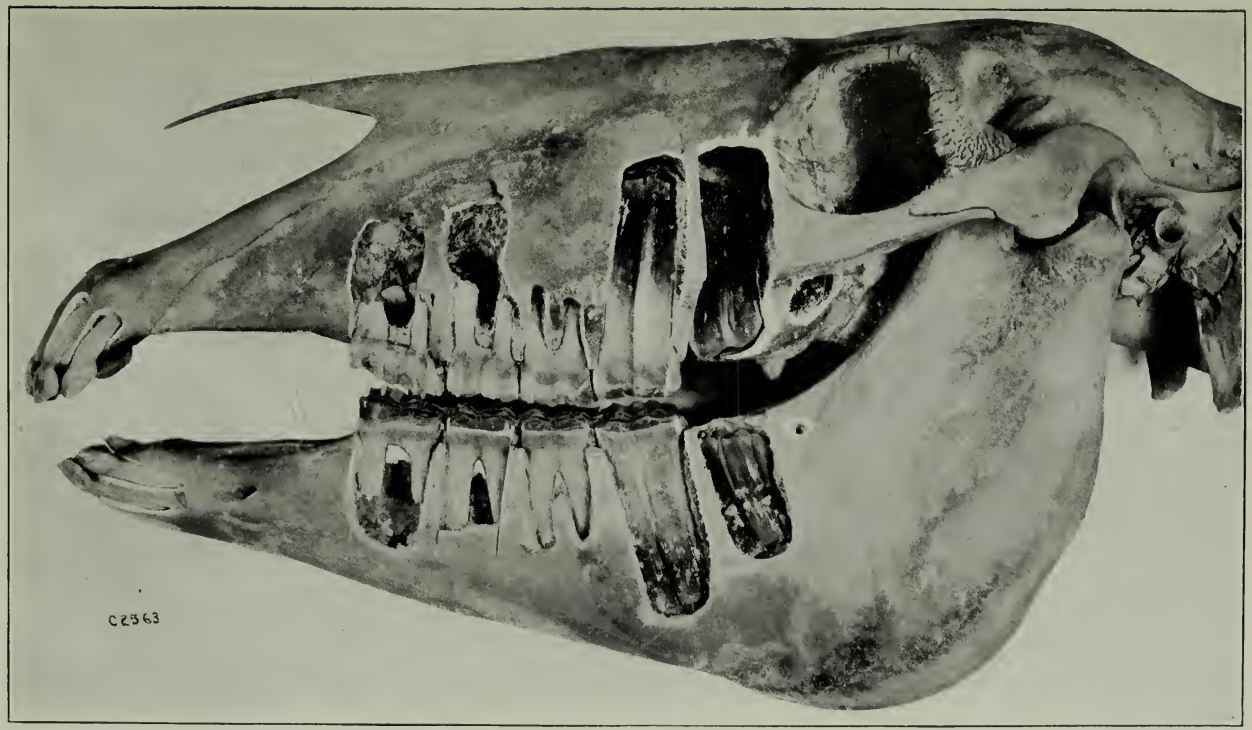

Fig. 68—Sculptured head of a 17-month colt.

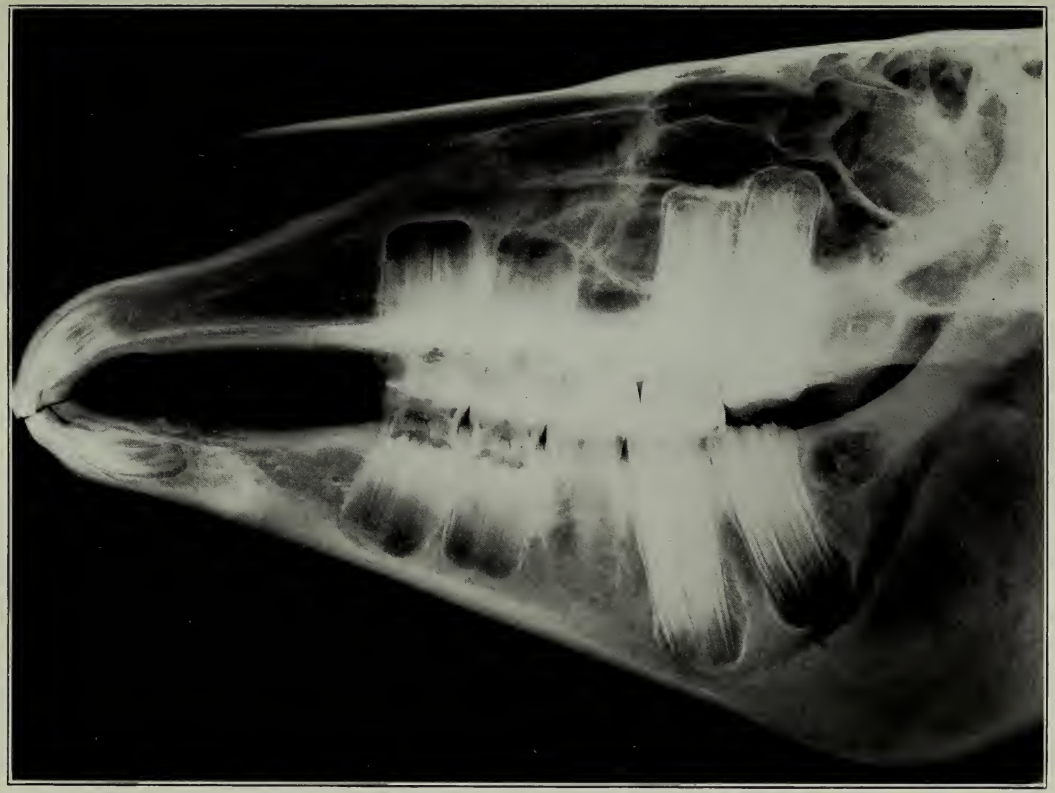

Fig. 69—Radiograph of a 23-month-old colt (underdeveloped).

Fig. 70—Radiograph of a 31½-month mare.

Fig. 71—Radiograph of a four-year ten-month stallion.

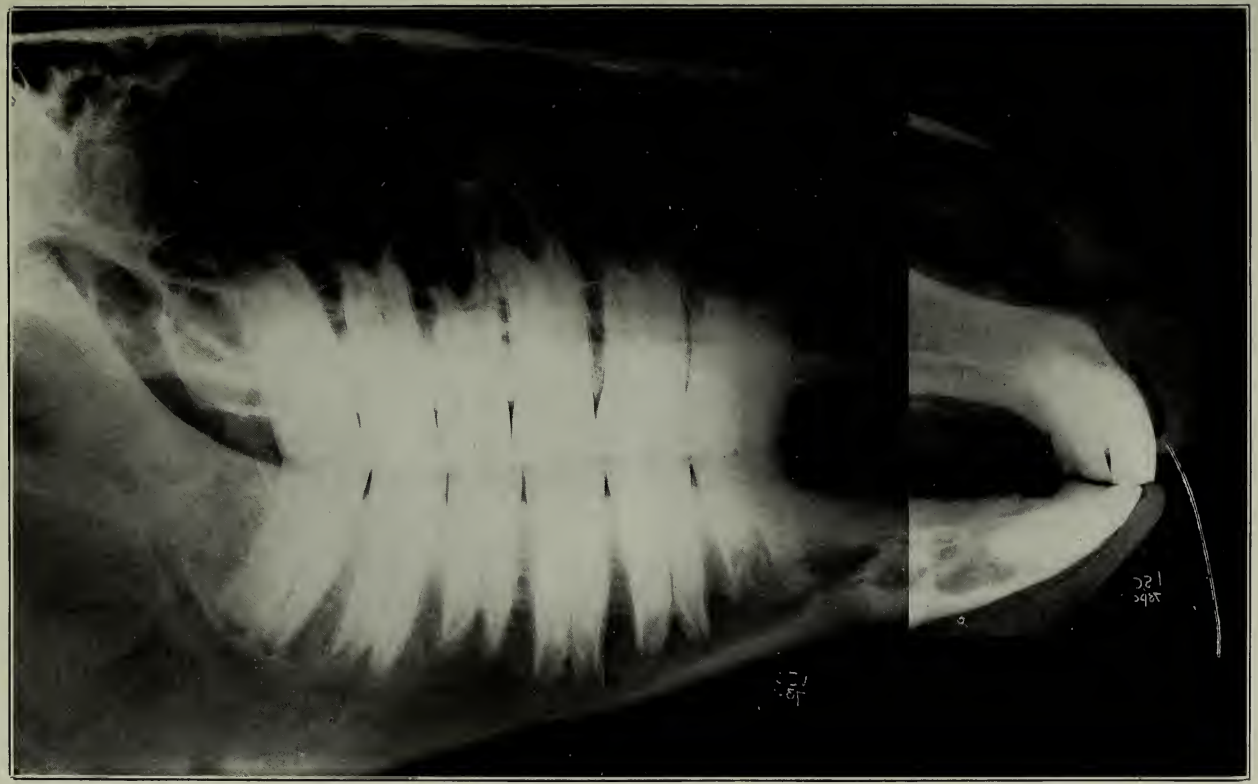

Fig. 72—Radiograph of a five-year eight-month eleven-day-old mare.

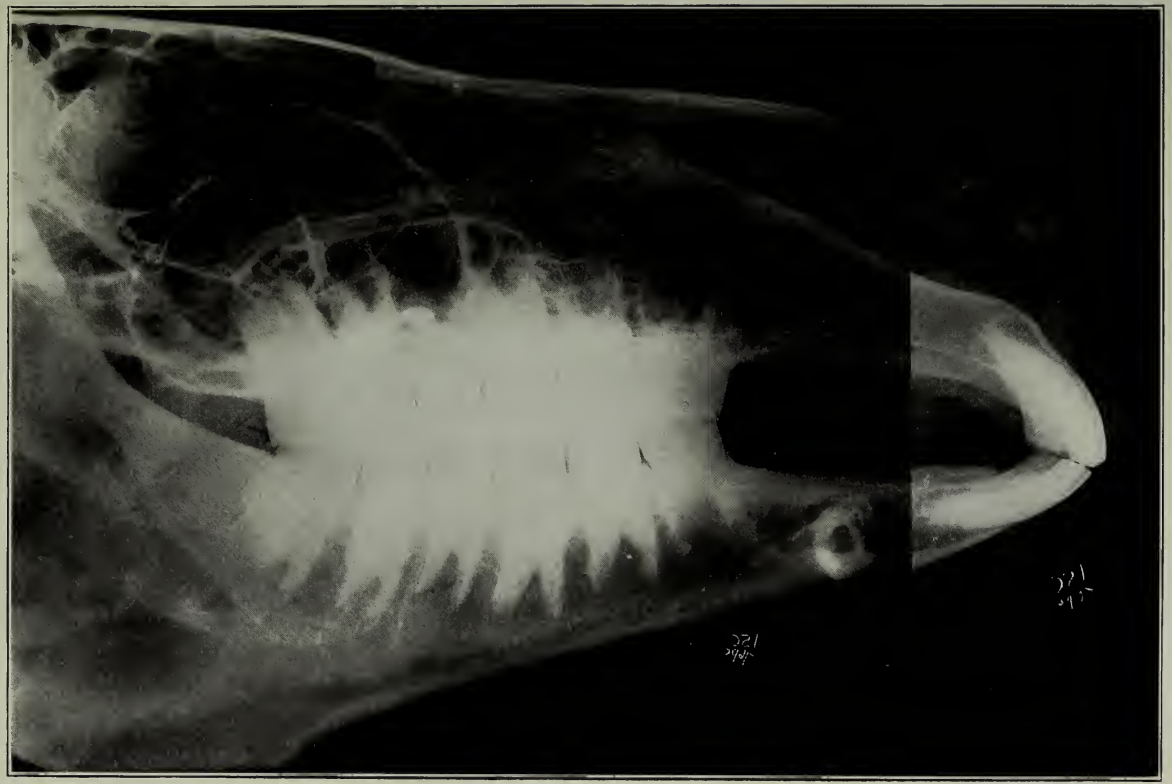

Fig. 73—Radiograph of a seven-year-old mare.

Fig. 74—Radiograph of a ten-year four-month hackney stallion.

Fig. 75—Radiograph of a twelve-year-old mule.

Fig. 76—Radiograph of a fourteen-year 14-day-old mare.

Fig. 77—Radiograph of a sixteen-year-old standard bred mare.

Fig. 78—Radiograph from an old horse to show naso-lacrimal duct injected with opaque material; the wires are in the infraorbital canal and the parotid duct.

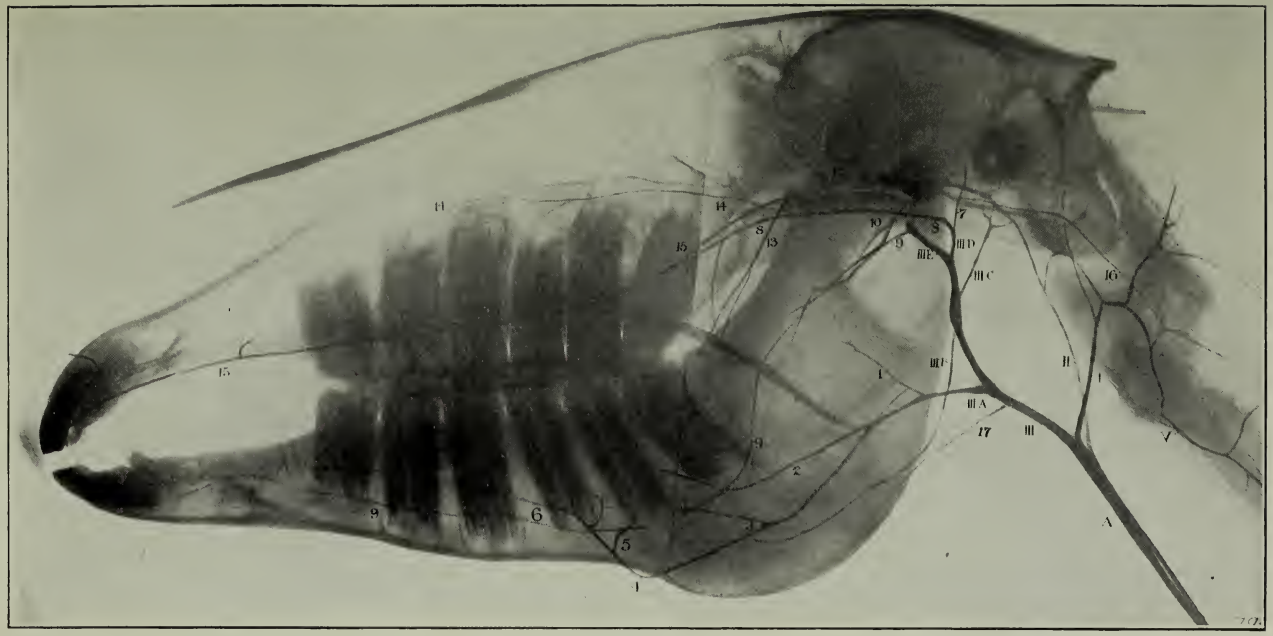

Fig. 79—Radiograph of vascular injection of head. A. Common carotid artery. 1. Occipital artery which anastomosis with the (v) vertebral on the dorsal aspect of the wing of the atlas. II. Internal carotid artery. III. External carotid artery. IIIA. External maxillary artery. 1. Pharyngeal artery. 2. Lingual artery. 3. Sublingual artery. 4. External maxillary winding around border of mandible to become the facial artery. 5. "The anterior masseteric artery." 6. Inferior labial artery. 6′. Superior labial artery (other branches not shown, see figure 47). IIIB. Masseteric artery ("posterior masseteric artery"). IIIC. Posterior auricular. IIID. The superficial temporal artery. 7. Anterior auricular artery. 8. Transverse facial artery. IIIE. Internal maxillary artery. 9. Inferior alveolar. 10. Pterygoid arteries. 11. Posterior deep temporal. 12. Anterior deep temporal. 13. Buccinator artery. 14. Infraorbital artery. 15. The greater palatine.

Fig. 80—Microphoto of a cheek tooth from a five-month fetus. 1. Oral epithelium. 2. Developing bone. 3. Pulp. 4. Infundibulum.

Fig. 81—Microphoto of cusps (crown) of 79. 1. Pulp. 2. Stellate reticulum of enamel organ. 3. Odontoblasts. 4. Adamantoblasts. 5. Dentin. 6. Enamel. 7. Blood vessel (engorged).

Fig. 82—Microphoto of a small portion of 81 to show detail of dentin, etc. 1. Pulp. 2. Odontoblasts. 3. Dentin. 4. Dento-enamel junction. 5. Enamel. 6. Adamantoblasts. 7. Stellate reticulum (of enamel organ within infundibulum).

Fig. 85—A supernumerary tooth (7th?) above.

Fig. 86—Supernumerary superior incisor posterior to left corner.

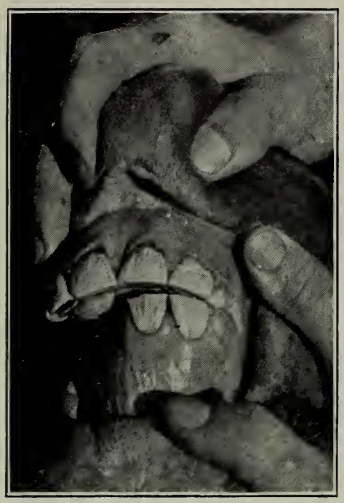

Fig. 83. Congenital absence of mandibular incisors of the right side.

Fig. 84—Inferior canine directly against the corner incisor.

Fig. 87—Supernumerary incisor posterior to right intermediate.

Fig. 90—Five incisors dorsal and ventral view (specimen of Dr. Gubser, Earlham, Iowa).

Fig. 88 — Retained "milk tooth" between left corner and intermediate, and malformed left third mandibular tooth, leaving space postero-medial to tooth for food accumulation and loss of antero-posterior alignment.

Fig. 89—Five incisors, and separation between right first and second mandibular teeth.

Fig. 91—Maxillary alveoli of an old horse to show root pattern.

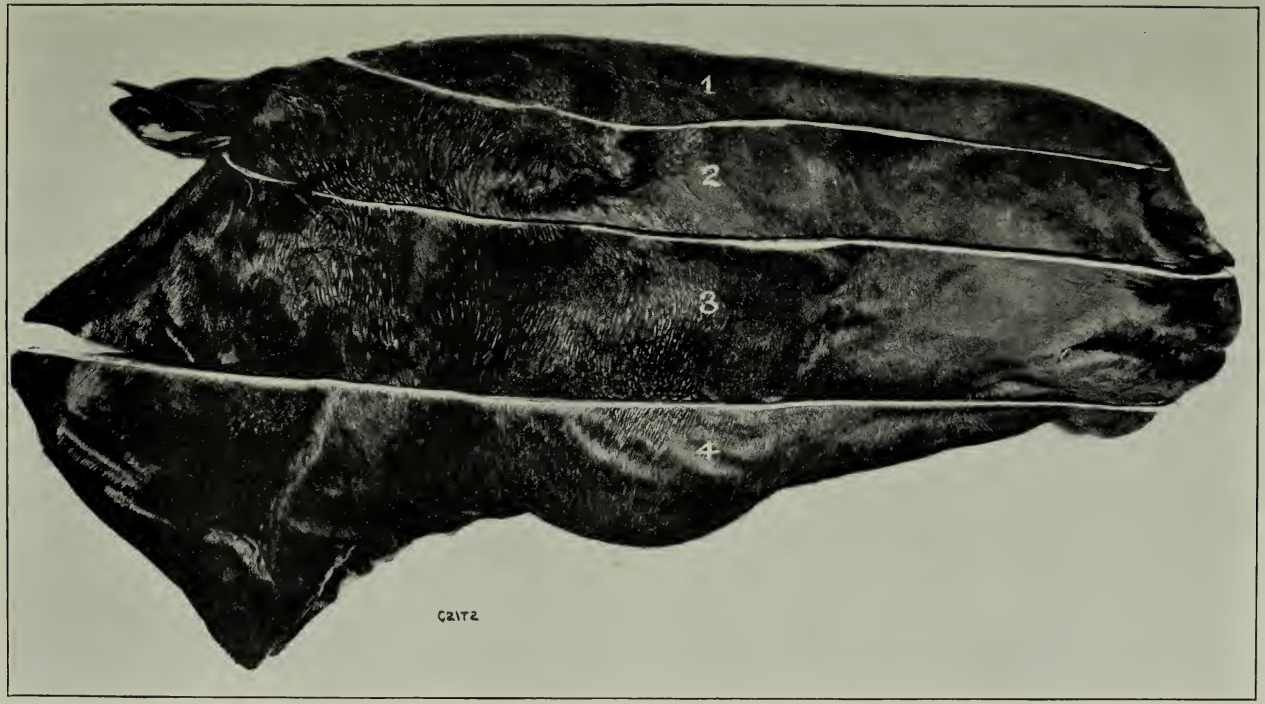

Fig. 92—Lateral view of head cut in horizontal section. See Figs. 91 to 98, inclusive. 1. Section No. 1. 2. Section No. 2. 3. Section No. 3. 4. Section No. 4.

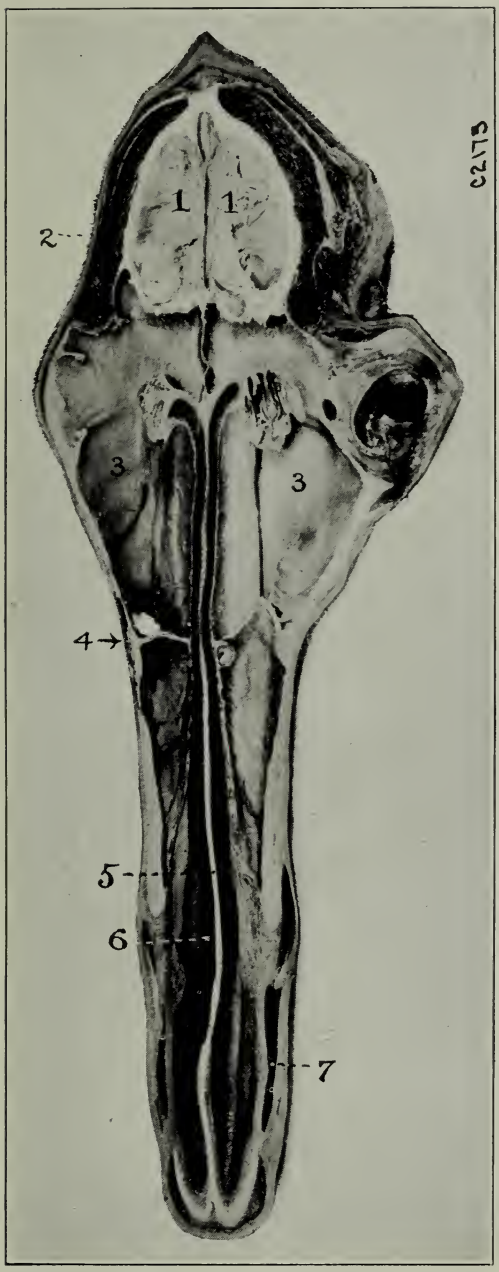

Fig. 93—Ventral view of Section 1. 1. Cerebrum. 2. M. Temporalis. 3. Frontal sinus. 4. Anterior wall of frontal sinus. 5. Septum nasi. 6. Common meatus nasi. 7. Diverticulum nasi.

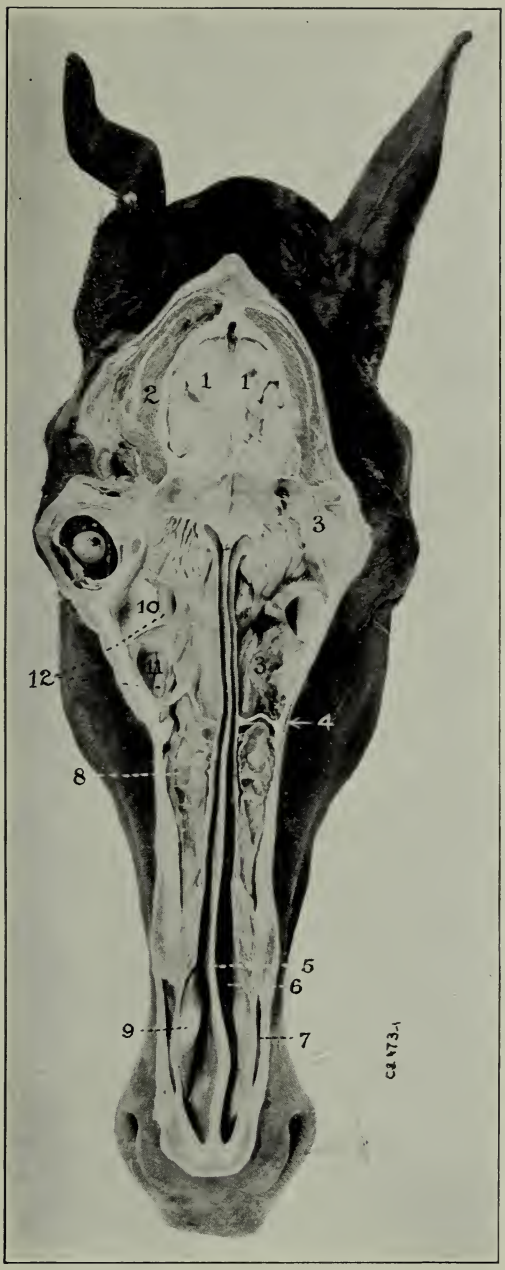

Fig. 94—Dorsal view of Section 2. 1 to 7. See Fig. 93. 8. Cavity of scrolled portion of superior turbinate bone. 9. Anterior cartilaginous portion of superior turbinate. 10. Fronto maxillary opening. 11. Anterior maxillary sinus (see text). 12. Infraorbital canal.

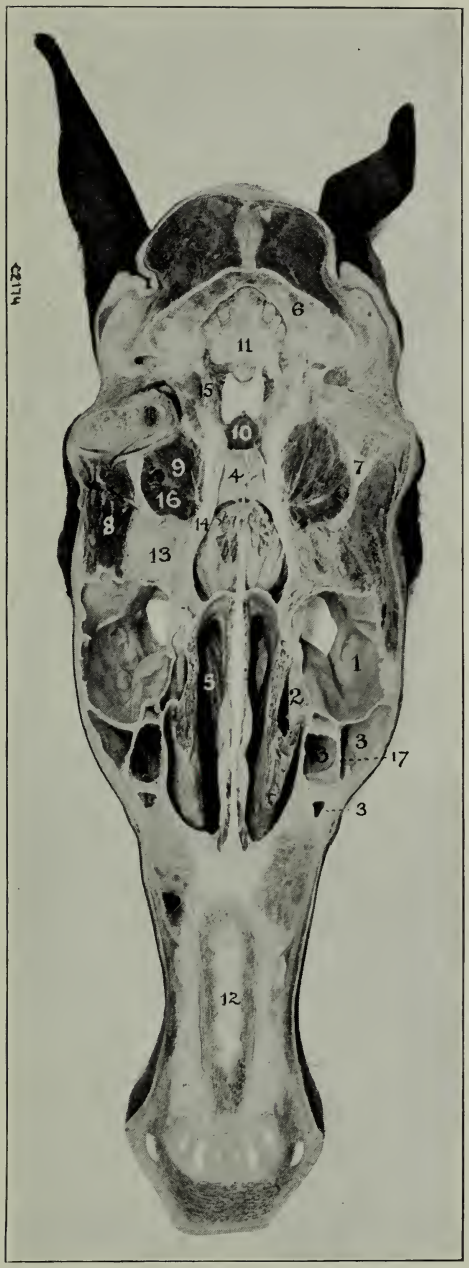

Fig. 95. Ventral view of Section 2. 1. Maxillary sinus. 2. Turbinal part of anterior maxillary sinus. (Figure directly on infraorbital canal.) 3. Lateral or maxillary part of anterior maxillary sinus. 4. Sphenopalatine sinus. 5. Common meatus of nasal cavity. 6. Occipital bone. 7. Mandible. 8. M. Masseter. 9. M. Pterygoideus lateralis. 10. Pituitary body. 11. Cerebellum. 12. Oral cavity (a small portion of mucous membrane of hard palate shown). 13. Periorbital fat. 14. Fifth cranial nerve. 15. Semi-lunar ganglion. 16. Temporalis muscle (lower part). 17. Sagittal plate of bone over lateral fangs of teeth.

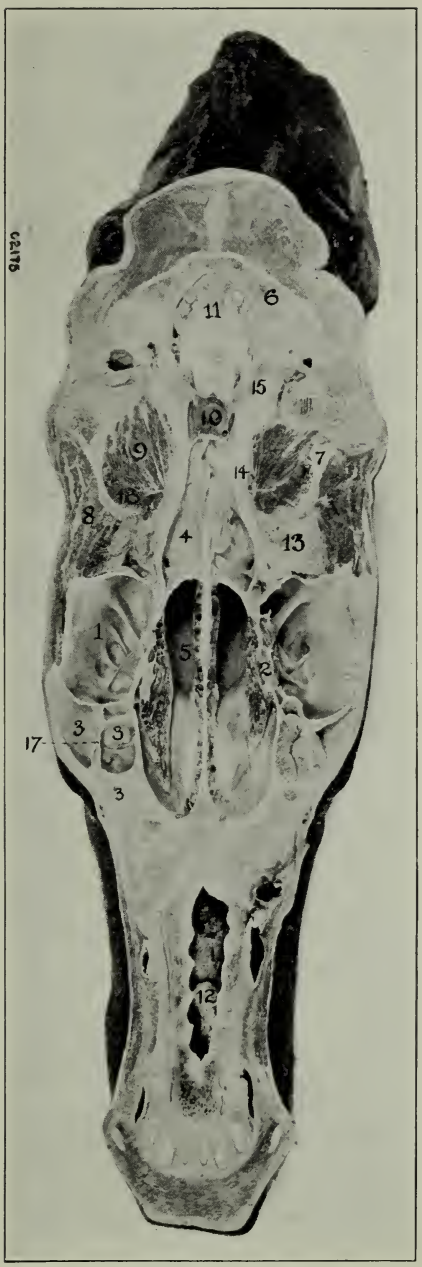

Fig. 96—Dorsal view of Section 3. For legend see Fig. 95. No. 12 is on dorsum of tongue within oral cavity.

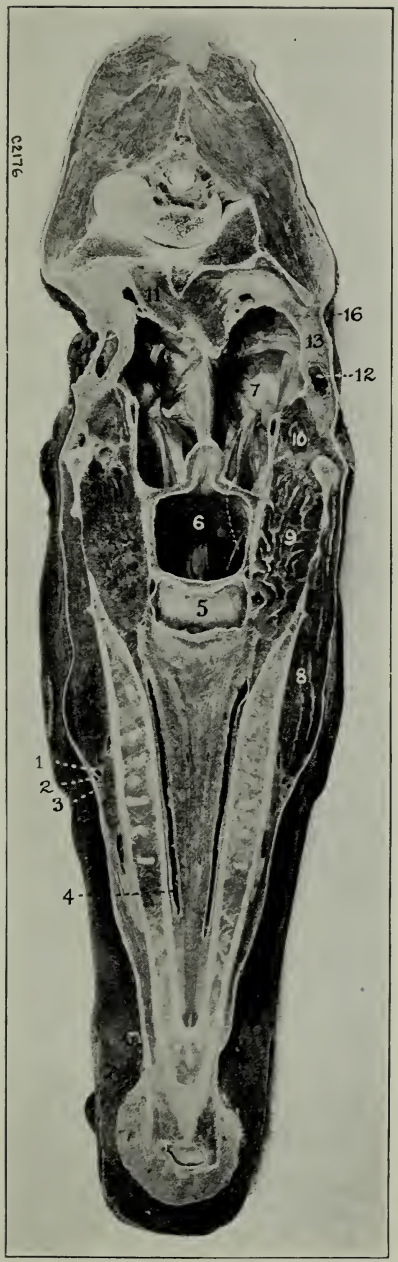

Fig. 97—Ventral view of Section 3. 1. Facial vein. 2. Parotid duct. 3. Facial artery. 4. Oral cavity. 5. Soft palate (cut edge above figure). 6. Pharynx: dotted line from eustachian orifice to groove (canal) in gutteral pouch. 7. Gutteral pouch. 8. M. Masseter. 9. M. Pterygoideus internus. 10. Mm. Digastricus et occipito-mandibularis. 11. Ventral straight muscles of head. 12. Jugular vein. 13. Parotid salivary gland. 16. Mandibular salivary gland.

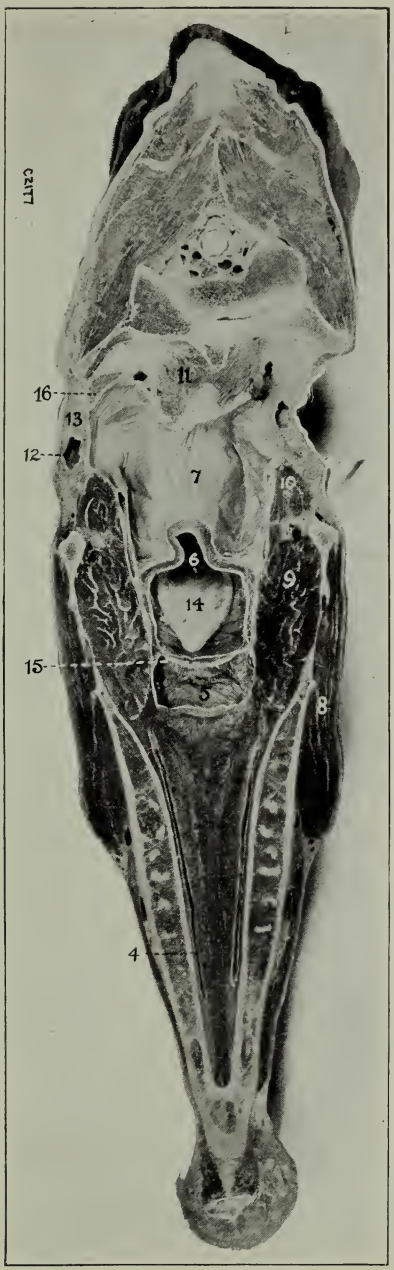

Fig. 98—Dorsal view of Section 4. For legend see Fig. 97. 5. On root of tongue in fauces. 14. Epiglottis. 15. Cut edge of soft palate (between oral cavity and pharynx).

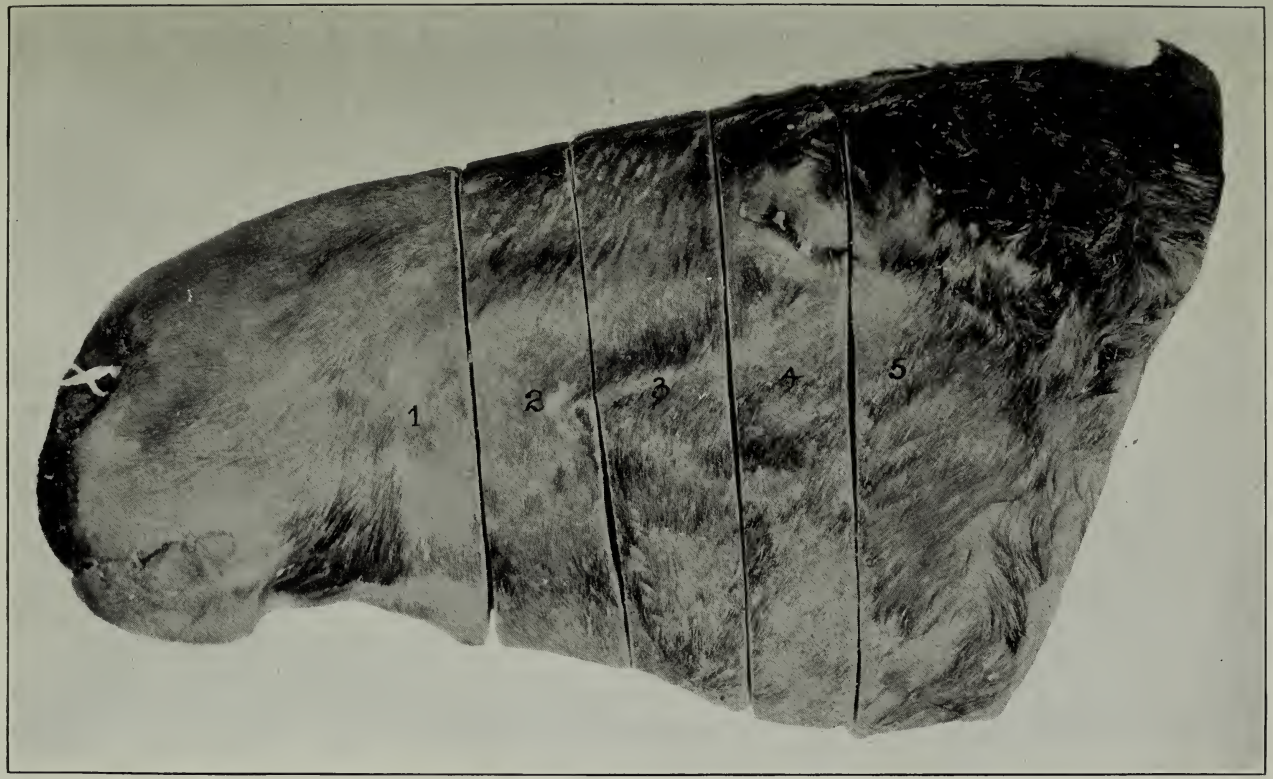

Fig. 99—Lateral view of a two-year-and-eight-month head cut in cross sections and assembled; sections numbered from before backward.

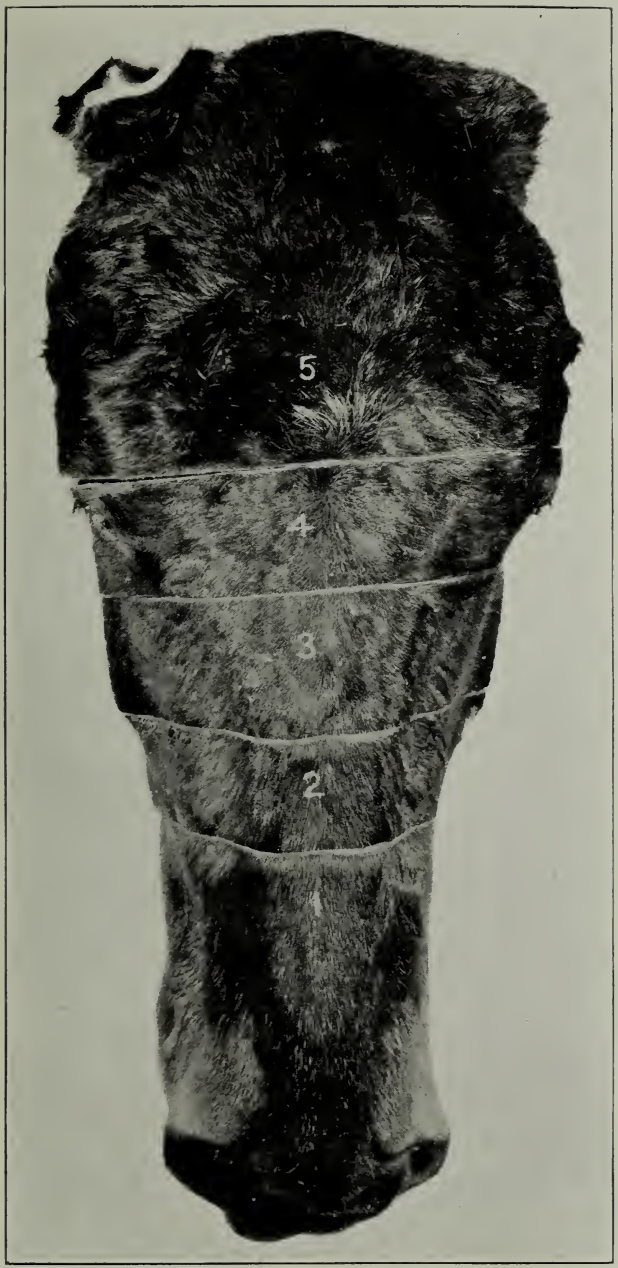

Fig. 100—Dorsal view of Fig. 99.

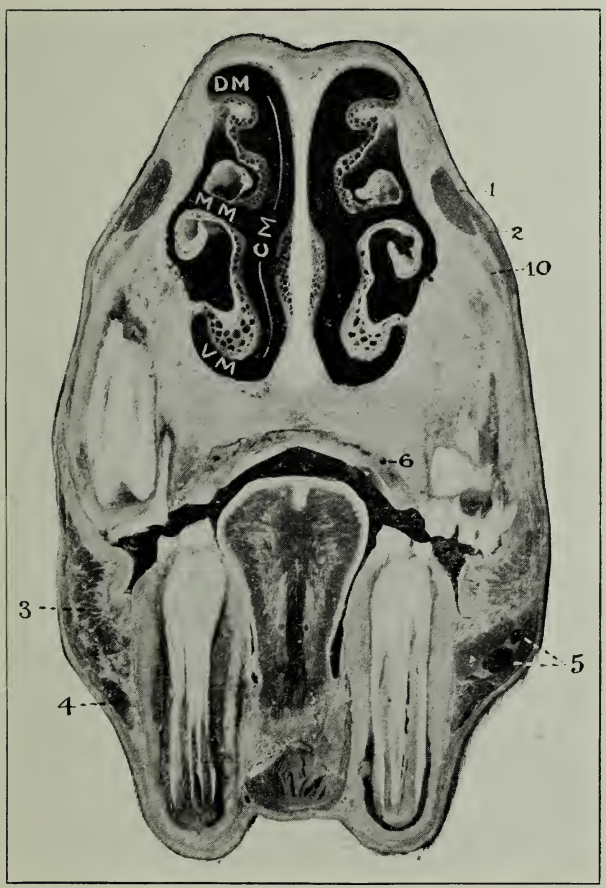

Fig. 101—Posterior view of Section 1. 1. M. Levator labii superioris proprius. 2. M. Levator naso-labialis. 3. M. Buccinator. 4. Depressor labii inferioris. 5. Buccal veins. 6. Palatine artery. 10. Infraorbital nerve. DM, dorsal meatus nasi. MM, middle meatus nasi. VM, ventral meatus nasi. CM, common meatus nasi.

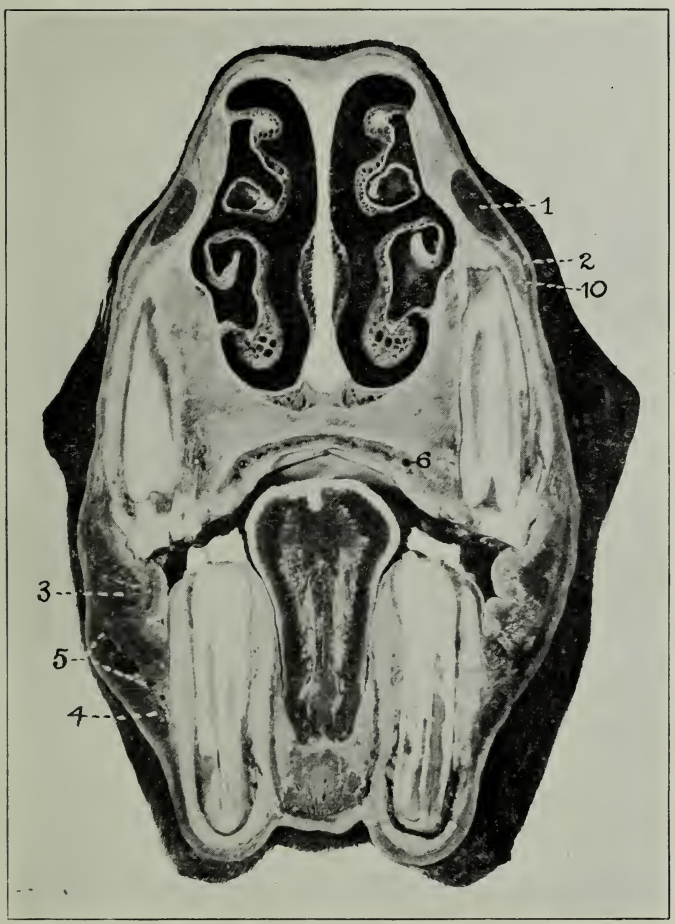

Fig. 102—Anterior view of Section 2. For legend see Fig. 101.

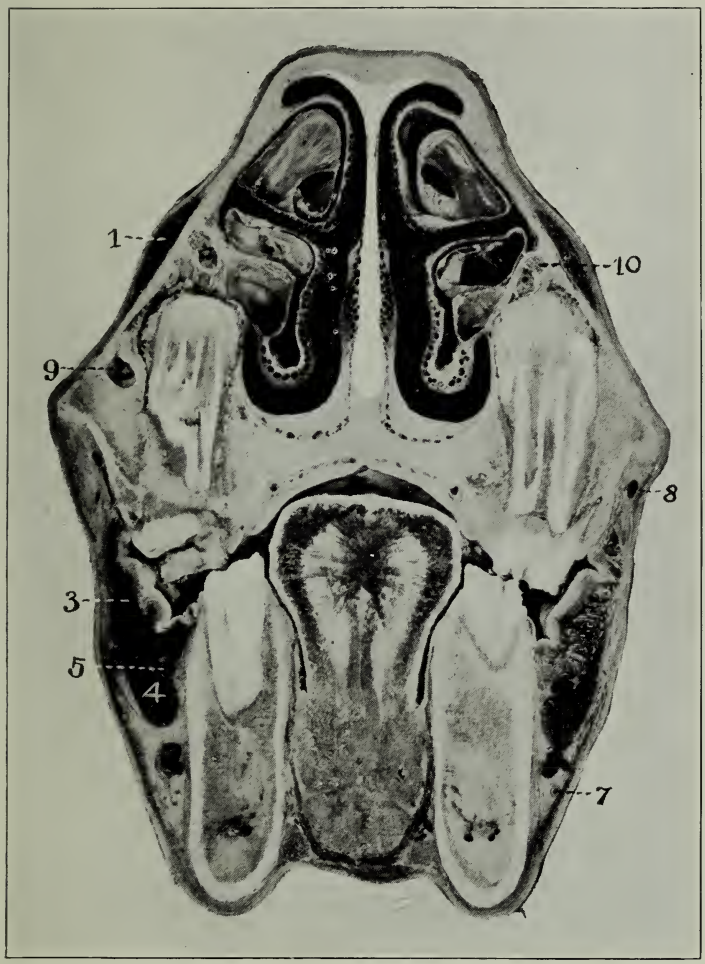

Fig. 103—Posterior view of Section 2. For 1, 3, 4, 5 and 10 see Fig. 101. 7. Inferior buccal artery. 8. Transverse facial vein. 9. Anterior part of lateral portion of anterior maxillary sinus. 10. Infraorbital nerve (naso-lacrinal duct ventro medially).

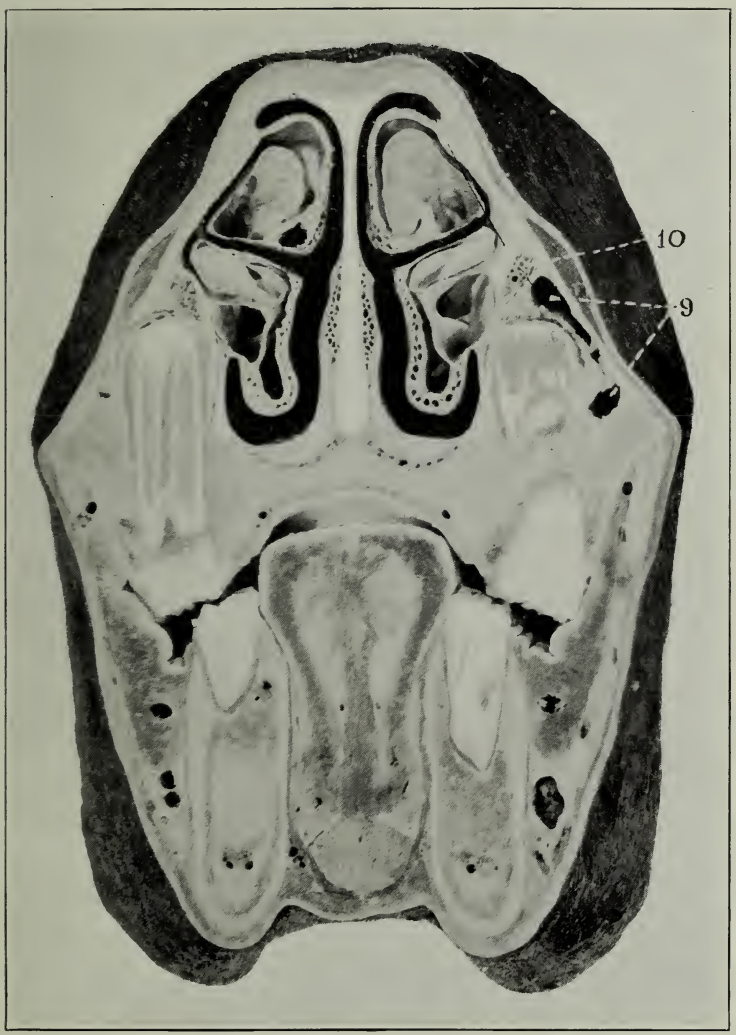

Fig. 104. Anterior view of Section 3. For legend see Fig. 103.

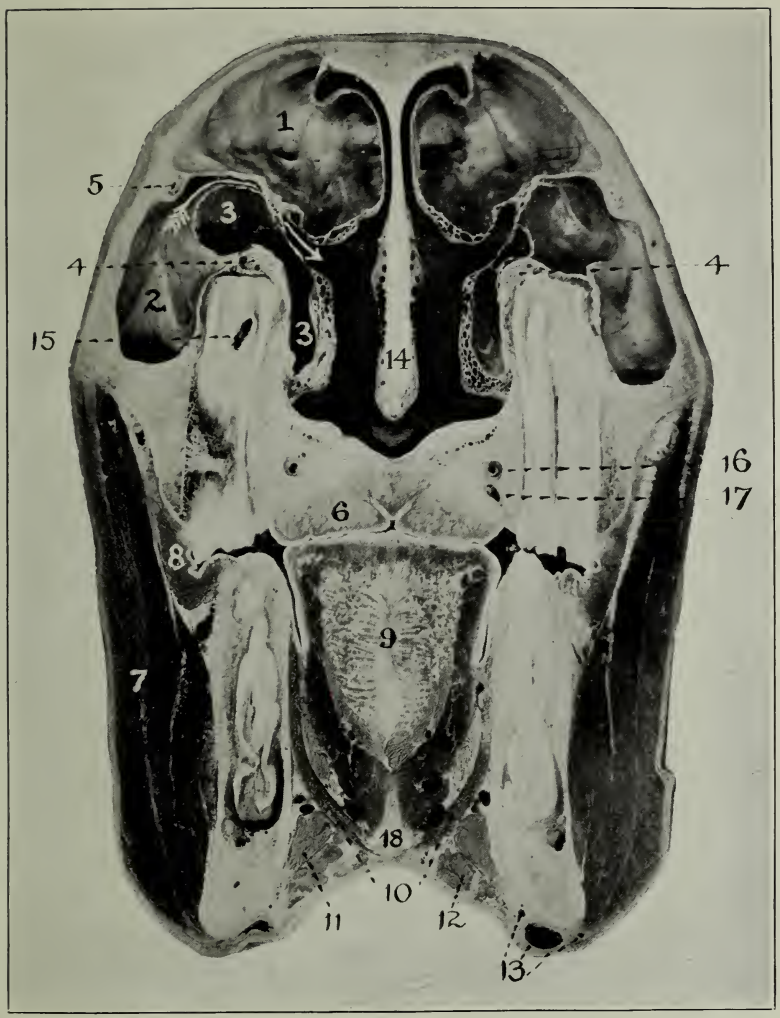

Fig. 105—Posterior view of Section 3. 1. Anterior part of frontal sinus (sinus portion of the superior turbinate). 2. Anterior part of posterior maxillary sinus, arrow in naso-maxillary fissure. 3. Anterior maxillary sinus. 4. Infraorbital nerve. 5. Naso-lacrimal duct. 6. Soft palate (glandular portion). 7. M. Masseter. 8. Mm. Buccinator and depressor labii inferioris. 9. Tongue. 10. M. Mylohyoideus. 11. M. Digastricus. 12. Mandibular lymph nodes. 13. Parotid duct and facial vessels. 14. Septal cartilage (just posterior to operative field; note vessels; see Fig. 104). 15. Incomplete cementation in bottom of infundibulum. 16. Palatine artery (within bony canal). 17. Palatine vein. 18. Lingual process of hyoid bone.

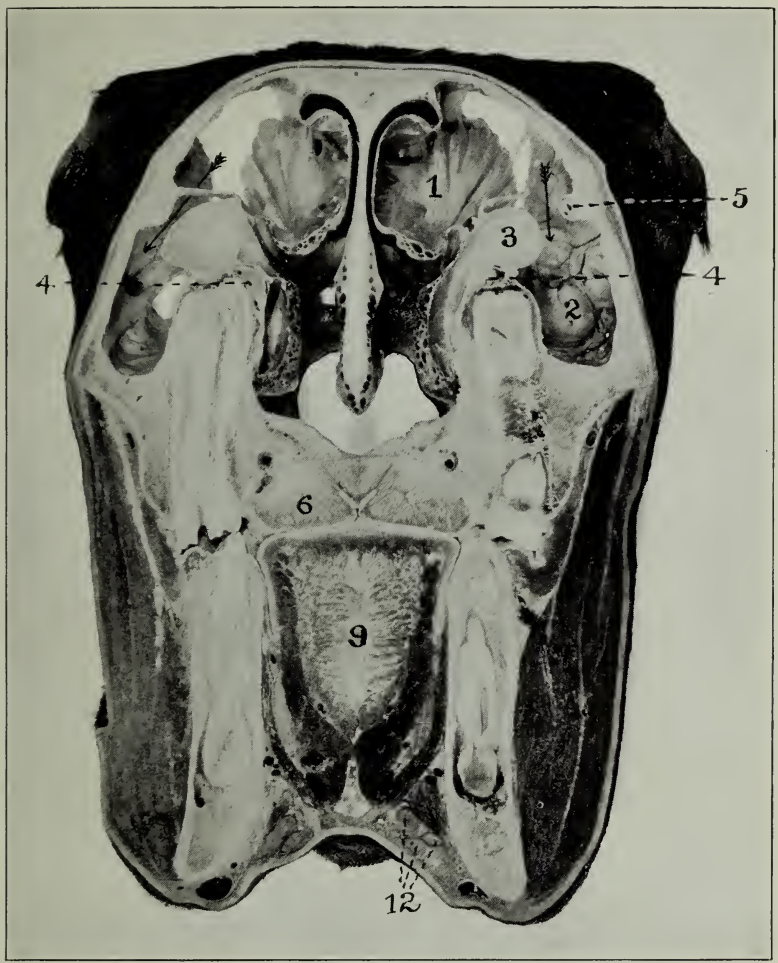

Fig. 106—Anterior view of Section 4. 1. Posterior part of turbinal portion of frontal sinus (figure on laminae lateralis of ethmoid bone). 2. Posterior part of posterior maxillary sinus: arrows are in the fronto-maxillary opening. 3. Posterior part of anterior maxillary sinus. 4, 5, 6, 9 and 12. See Fig. 105.

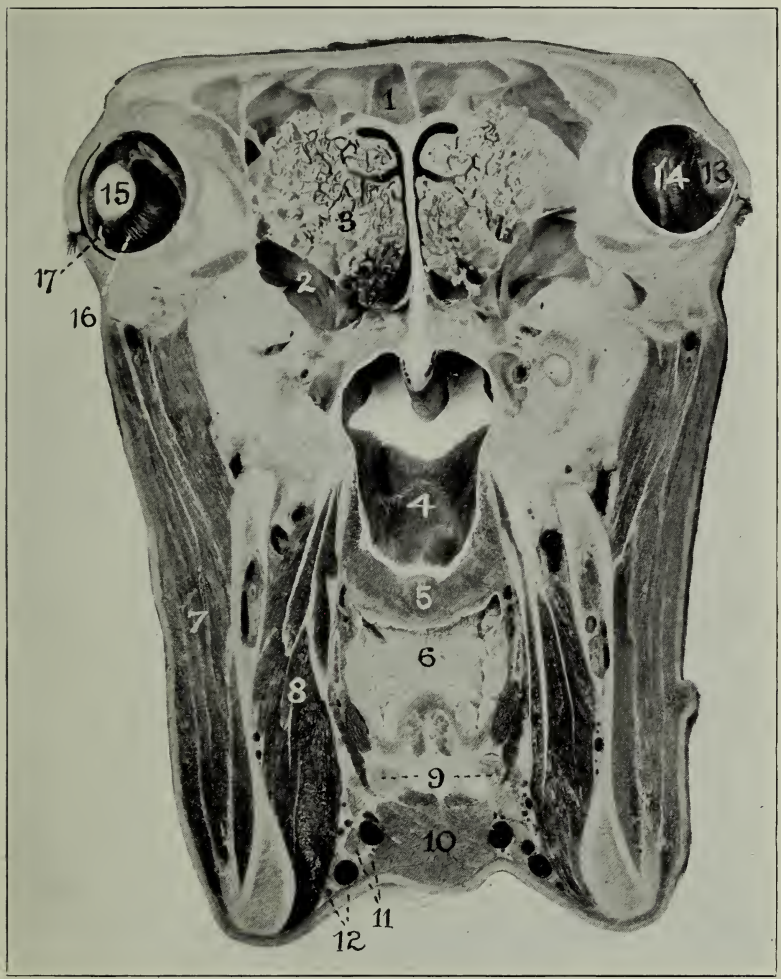

Fig. 107—Posterior view of Section 4. 1. Frontal sinus. 2. Posterior maxillary sinus. 3. Lateral masses of ethmoid bone and their meati. 4. Pharynx. 5. Cut edge of soft palate. 6. Fauces (note the glands in the mucous membrane ventrally and laterally). 7. M. Masseter. 8. M. pterygoideus internus. 9. Small cornu of the hyoid bone. 10. Mm. Omohyoideus and hyoid portion sterno thyro-hyoideus. 11. Mandibular lymph nodes. 12. External maxillary vessels. 13. Anterior compartment of eye. 14. Posterior compartment of eye. 15. Crystalline lens. 16. Ciliary processes. 17. Iris.

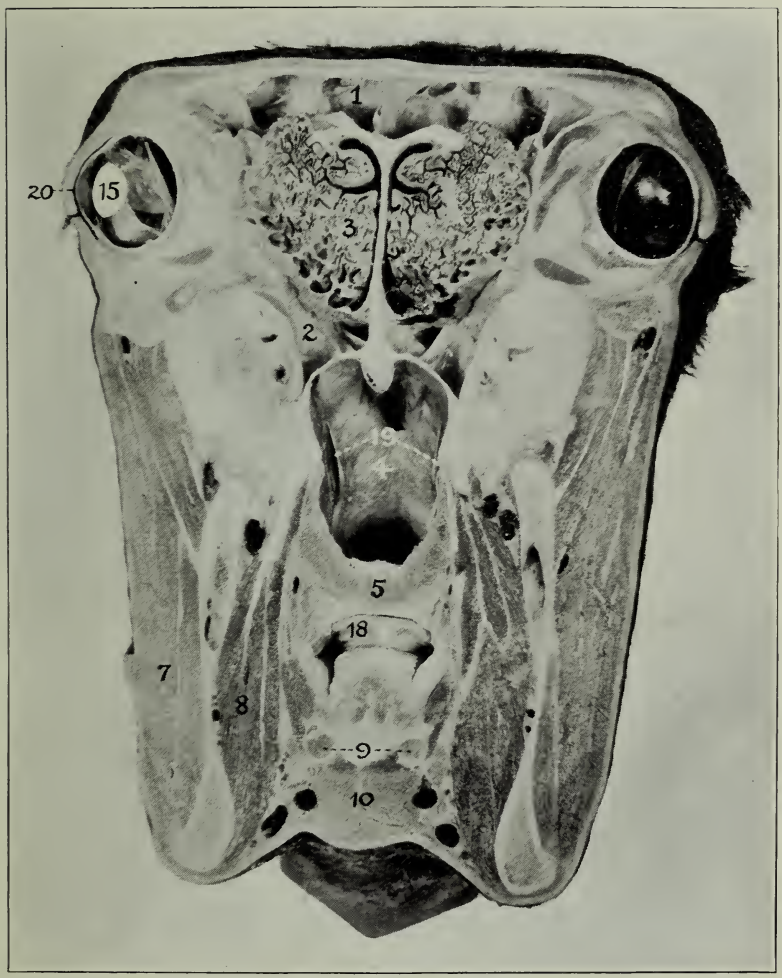

Fig. 108—Anterior view of Section 5. For legend see Fig. 107. 18. Epiglottic cartilage. 19. Eustachian orifices. 20. Conjunctiva.

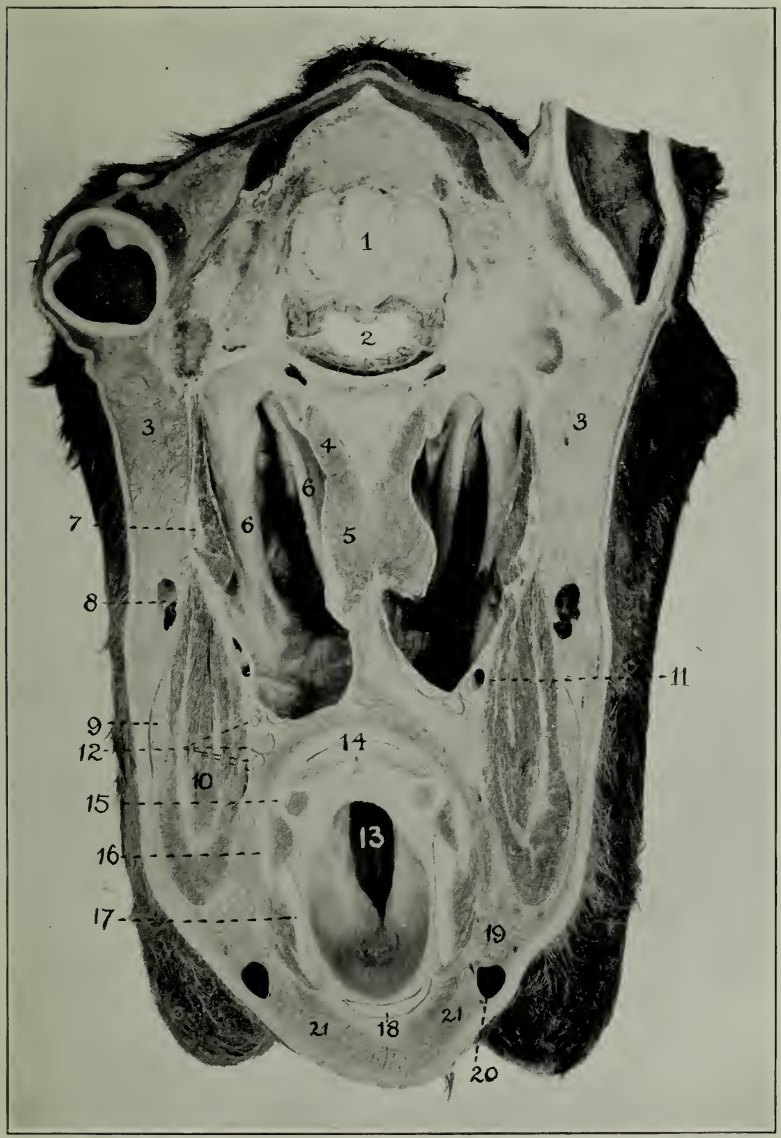

Fig. 109—Posterior view of Section 5. 1. Cerebellum. 2. Medulla ablongata. 3. Parotid salivary gland. 4. M. Rectus capitis ventralis minor. 5. M. Rectus capitis ventralis major. 6. Gutteral pouch. 7. Great cornu of hyoid bone. 8. Jugular vein. 9. Tendon of M. sterno cephalicus. 10. M. Occipito-mandibularis. 11. External carotid artery. 12. Pharyngeal (para pharyngeal) lymph nodes. 13. Rima Glottidis. 14. Esophagus. 15. Arytenoid cartilage. 16. Lamina of thyroid cartilage. 17. Cricoid cartilage. 18. First ring of trachea. 19. Mandibular salivary gland. 20. External maxillary vein. 21. Mm. Omohyoideus and hyoid portion of steron thyro-hyoideus.

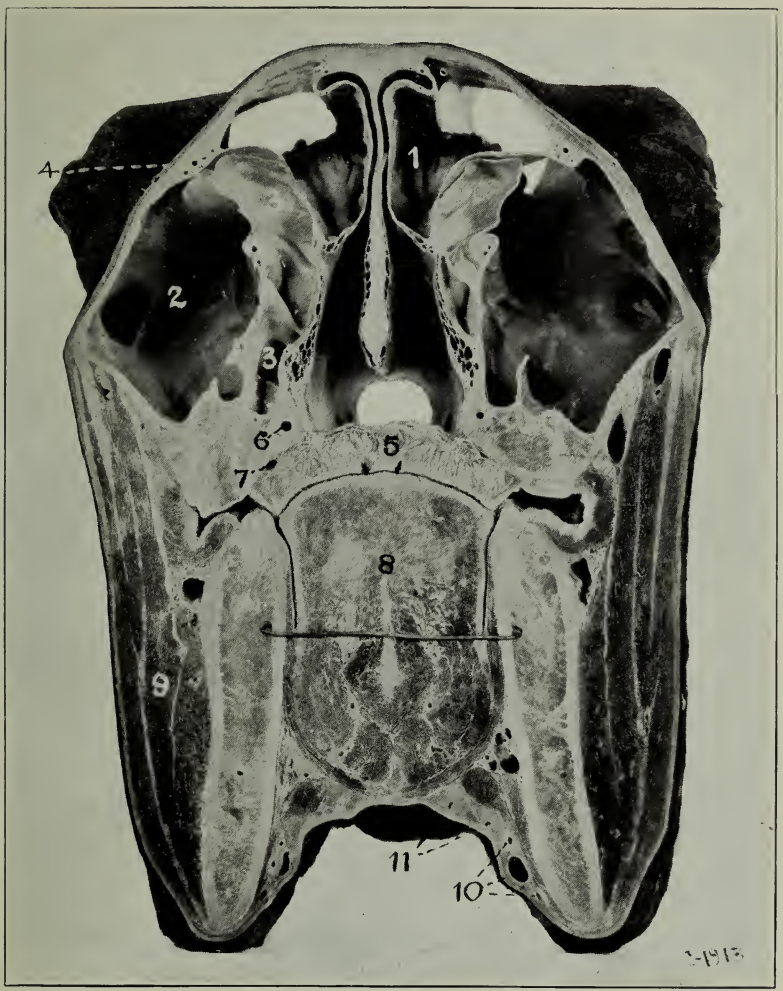

Fig. 110—Anterior view of a cross-section of an old horse (this section is the one between Fig. 108 and 107 first report which see). 1. Frontal sinus. 2. Posterior maxillary sinus. 3. Anterior maxillary sinus. 4. Naso-lacrimal duct. 5. Soft palate (glandular portion). 6. Palatine artery (within bony canal). 7. Palatine vein. 8. Tongue. 9. M. Masseter. 10. Parotid duct and external maxillary vessels. 11. Mandibular lymph nodes.

PHYSIOLOGY OF DIGESTION IN HERBIVORA

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Gentlemen: It has been said that good physiology is the best preventive of bad medicine. In other words, a thorough understanding of the normal functions of the body organs and systems, will enable easier recognition of abnormal or deranged functions, and hence, lead to greater accuracy in diagnosis and in the treatment applied to assist in the restoration of the diseased tissues. The foundation for rational therapeutics is a proper understanding of the normal body functions. The animal body is a complex of a large number of different organs, differing in their histological structure, in keeping with their functional characteristics. It is not only essential that a general understanding of the functions of all these organs be attained by the practitioner, but a more detailed knowledge of the functions of those more frequently diseased must be acquired. Normal variations must be recognized, that is, the varying intensity with which each function may be performed under different circumstances. Of all the varying conditions, whose influence on the functions of the organs must be taken into consideration, there is none so important as the action of the organs themselves upon one another, and the consequent great interdependence among them. A knowledge of this interdependence is an important factor in rational therapeutics, for in a great number of instances drugs are not given to act particularly upon the diseased tissues, but upon healthy ones, for an abnormal condition in the case of one organ causes changes in the interrelations of other body organs, and possibly may bring about a general functional incoordination. This functional interdependence among the body organs is particularly noticeable in discussing the subject to be considered at this period, i. e., the physiology of digestion, for it will be shown that often the functional activities of certain organs and glands of the digestive tract are dependent upon certain substances formed in other organs and carried to them.

Before proceeding with the special subject assigned for this period, it might be well to call attention to the remarkable progress made by the science of physiology in the past few years. It is to be granted that some of the phenomena of life, such as physiology attempts to interpret, can only be explained by theoretical deduction, however, this no longer need go further than similar daily methods of the practitioner, who interprets correctly diseased conditions by processes which he cannot see. Physiology now makes use of all the modern aids to re-

search in natural science, employing the finest instruments of precision, and highest mathematical analysis. It would be a hopeless task to take up in any detail an account of the multifarious methods employed in the experimental investigations, even of the subject under consideration this morning, hence during the discussion a description of a few of the typical methods only must suffice. The speaker will be glad to explain in greater detail at any time regarding these experimental methods, and demonstrate some of the newer apparatus for physiological experimentation which may be seen in the laboratory.

In the animal body the conversion of the materials taken as food into an assimilable form is known as digestion and is carried on in the alimentary canal, chiefly by the actions of a variety of enzymes secreted into the canal by the various glandular organs opening into it, or by glands located within its walls. Bacteria also play an important part in the digestion of certain foodstuffs, this being especially noticeable in cellulose digestion in herbivora. To describe the great variety of digestive mechanisms met with in the animal kingdom would consume much more than our allotted time. In all cases, whatever the nature of the digestive mechanism may be, the object is to insure the effective action of the digestive enzymes, and the due absorption by the blood or lymph of the products of their activity.

In discussing the physiology of digestion in herbivora this morning, especial emphasis will be laid on the process as it occurs in ruminants, inasmuch as this type is the subject of the discussion for the day. However, contrasts will be drawn with the processes in the horse which are fundamentally the same as in the ox, certain of the processes merely occurring at opposite ends of the alimentary canal. No attempt at detail will be made on account of the limited time, but rather a general review, emphasizing certain of the newer facts, the results of research in the past few years, and endeavoring to bring out certain points of practical advantage to the practitioner. The following general outline will be followed:

A. THE HYDROLYTIC ENZYMES OF THE ALIMENTARY CANAL.
B. DIGESTION IN THE MOUTH.
   a. Prehension of Food.
   b. Mastication.
   c. Insalivation.
C. DEGLUTITION.
D. DIGESTION IN THE STOMACH.
   a. The Stomach Glands and Secretions.
   b. Stomach Digestion in the Horse.

c. Stomach Digestion in Ruminants.
d. The Gases of the Stomach.
e. Rumination and Vomition.
f. The Stomach Movements.

E. INTESTINAL DIGESTION.

a. The Pancreatic Secretion.
b. The Bile.
c. The Intestinal Glands and Succus Entericus.
d. Intestinal Digestion in the Horse.
e. Intestinal Digestion in Ruminants.
f. The Intestinal Movements.
g. Gases of the Intestine.

F. ABSORPTION OF THE FOODSTUFFS.

G. THE FECES AND DEFECATION.

THE HYDROLYTIC ENZYMES OF THE ALIMENTARY CANAL

The digestive processes involve both chemical and mechanical changes. The mechanical changes are brought about by the crushing and grinding undergone in the mouth, together with the mixing and kneading that come from the peristaltic movements of the stomach and intestines. The chemical changes are of a peculiar character, being affected through the agency of a group of bodies known as enzymes or ferments, which are contained in the various secretions poured into the mouth, stomach, and intestines, or these enzymes may remain localized in the walls of certain of the digestive organs. The enzymes are the essential factors in the various secretions which produce the chemical changes in the foods. Enzymes cannot be produced by chemical methods. They are found only as the products of the living protoplasm of cells, and a distinction is sometimes made between endo or intra cellular enzymes which are not secreted but exert their action within the cells in which they are contained, and exo or extra cellular enzymes which are eliminated from the cells in which they are formed and are found in solution in the various secretions. The ptyalin of the saliva, or the pepsin of the gastric juice are examples of this latter class which has been studied more thoroughly than the first class, as many of these glandular secretions, by following the methods of Pawlow, Bayliss and Starling, may be collected in their pure form and with them the enzymes.

Enzymes act by catalysis, that is by their presence they facilitate chemical reactions without supplying any energy to the reaction, without being used up, and without taking any part in the formation of the end products. Minimal quantities will induce chemical changes involving almost indefinite quantities of other substances, the quantity of enzyme determining only the rapidity of the action, not the amount; a small quan-

tity will digest as much as a large quantity, but it will take a longer time. The digestive enzymes, with the exception of possibly those of the small intestine, exist within the gland cells in what is termed a zymogen or proferment form. The zymogen may be stored in the cell in the form of granules, which are converted into active enzymes at the moment of secretion, or it may be secreted in an inactive form and require the co-operation of some other substance, before it is capable of affecting its normal reaction. In such cases the second substance is said to activate the enzyme, and if it is an inorganic substance that causes the activation it is simply designated as an activator, while organic substances serving the same role are termed kinases. The enzymes playing the chief parts in the digestive processes all act by hydrolysis, that is, they cause the molecules of the substance acted upon to undergo decomposition by a reaction with water, hence they are termed hydrolytic enzymes. Thus in the conversion of the disaccharide sugar maltose to dextrose by the enzyme maltase in the small intestine, the reaction may be expressed:

$$C_{12}H_{22}O_{11}+H_2O=C_6H_{12}O_6+C_6H_{12}O_6$$

maltose water dextrose dextrose

Some further interesting facts regarding enzymes are that each enzyme has an optimum temperature for its activity, even being destroyed by high temperature but usually not by low temperature, some act only in an acid medium, others in alkaline, and still others in either acid, alkaline, or neutral media. They are hindered by the accumulation of the products of their activity, and most of them cease acting altogether when these products reach a certain concentration, but will begin acting again on the removal of these products, or if the mixture be simply diluted.

Enzymes are generally specific in their action, that is an enzyme which can cause cleavage of the starch molecule will not act on fat or protein, or even upon other members of the starch group. A recognized phenomenon of enzyme action which has done much to explain the utilization by the tissues of the end products of digestion after absorption, is what is termed the reversible action of enzymes. The action of most enzymes is reversible, that is, the enzyme is not only capable of breaking up the molecule into its simpler end products, but is capable of taking these end products and resynthesizing them into a form similar to the original. The presence of endo enzymes in the various body tissues is undoubtedly for the purpose of building up fat, protein, etc., from the end products of digestion brought to them by the blood.

Kastle and Loevenhart in experiments with the enzyme lipase, which in the body acts upon neutral fats, converting

them to fatty acid and glycerin, a step that takes place in the digestion and absorption of fats, demonstrated that this enzyme would cause not only a hydrolysis of the substance acted upon, but also a synethesis of the split products again into fat. In the subcutaneous tissues, therefore, fat may be stored, or if the conditions are altered, the fat may be split to fatty acid and glycerin and be oxidized in the body as food. A similar reversibility has been shown for some of the other enzymes of the body, but whether or not all of them possess this power under the conditions of temperature, etc., that prevail in the body has not yet been thoroughly worked out.

In connection with the discussion of enzymes, it should be mentioned that there are substances formed in certain of the animal tissues which possess the property of inhibiting the activity of enzymes. Anti enzymes have been demonstrated for several of the digestive enzymes. According to Weinland and other observers the reason why the stomach does not digest itself, is that during life there is present in the mucous membrane of the stomach an anti enzyme (anti-pepsin), which inhibits the action of the proteolytic enzyme pepsin. A similar substance (anti-trypsin) is present in the intestinal mucosa, as well possibly as in the tissues of intestinal parasites, which would certainly be digested otherwise under the powerful influence of the proteolytic enzyme trypsin.

Partial List of Enzymes Concerned in the Processes of Digestion and Nutrition

	Enzyme	*Where chiefly found*	*Action*
Amylolytic	Ptyalin	Saliva	Converts starch to maltose.
	Amylase	Pancreatic secretion	Converts starch to maltose.
	Liver diastase	Liver	Converts glycogen to dextrose.
	Muscle diastase	Muscles	Converts glycogen to dextrose.
	Invertase	Small intestine	Converts cane sugar to dextrose and levulose.
	Maltase	Small intestine, salivary and pancreatic secretions	Converts maltose to dextrose.
	Lactase	Small intestine	Converts lactose to dextrose and galactose.
Lipolytic	Lipase (steapsin)	Pancreatic secretion, fat tissues, blood, etc.	Splits neutral fats to fatty acids and glycerine.
Proteolytic	Pepsin	Gastric juice	Converts proteins to peptones and proteoses.
	Trypsin	Pancreatic secretion	Converts proteins into simpler crystalline products.
	Erepsin	Small intestine	Converts peptones and proteoses into simpler products.
Coagulating	Rennin	Gastric juice	Coagulates milk.
Activating	Enterokinase	Small intestine	Converts trypsinogen to trypsin.
Deamidizing	Guanase		
	Adenases		
	Oxidases		
	Catalase		
	Arginase		

Digestion in the Mouth

In the way of a general remark in discussing the digestive processes proper, it must be emphasized that the appetite itself and the palatability of the food are of great and deep-seated importance in the activity of the digestive apparatus, for it will be seen that only under their influences does a plentiful secretion of saliva and gastric juice take place immediately upon the taking of food. The acid of the gastric juice in turn arouses the secretion of the pancreas, etc., when after a varying length of time, the stomach is emptied, the intestine is immediately prepared to continue the work of digestion and carry it forward to the end. Our knowledge of the influence of appetite upon the movements of the alimentary canal is somewhat meager. Certain recent observations by Carlson on man and dogs have shown without doubt that the sensation of hunger is attended with characteristic contractions of the empty stomach (hunger peristalses).

Prehension of Food: The methods of prehension of food vary with the species. The mobility and strength of the lips of the horse make them an important adjunct particularly in manger feeding, while in the ox the lips are rigid and the tongue with its horny papillae serves the purpose of grasping the food. In the sheep and goat class the divided upper lip allows of the incisors and dental pad biting close to the ground in grazing, and for this reason these animals can live on pastures where the horse and ox could not subsist. The capabilities for close grazing as possessed by sheep will often do serious damage to a good pasture. The ox also is better adapted, not only for the grasping and handling in the mouth of rougher and coarser food than the horse, but also for their further digestion.

Mastication: The most important function of the mouth with reference to digestion is the mechanical reduction of the food and the admixture of saliva with it, termed respectively mastication and insalivation, the movements of the tongue assisting markedly in both of these processes. The act of mastication is voluntary. Thorough mastication is essential for the subsequent solution and chemical transformation of the food, because the finer the state of division of the food, the larger the surface presented to the action of the digestive fluids, thus enabling them to exert their respective actions more effectively and in a shorter period of time. Mastication in the horse is a slow and thorough process, although there are individual variations. The tendency for digestive disturbances in "greedy" feeders, or often following the "bolting" of oats, are well known to the practitioner.

In the ox the first mastication is three times as rapid as in the horse and is of course imperfectly performed. Remastication is slow and thorough. The movements of the jaws in the horse and ox are not only up and down, but also lateral, and according to some observers backward and forward. Herbivora can masticate only upon one side at a time, during which time the salivary gland on that side secretes three or four times as much saliva as its fellow. In herbivora where lateral or rotary movement is necessary for grinding the food, the upper jaw is always wider than the lower, which peculiarity along with the oblique tables of the molars, tends to greater wear on the inside of the upper molars, and on the outside of the lower molars, which explains why the teeth of the horse and ox both become sharp on the inner and outer edges of the lower and upper molars respectively. The loss of condition and digestive disorders in animals due to improper mastication as a result of abnormal conditions associated with the teeth as well as the local irritation in the mouth are facts not to be overlooked.

Insalivation: During mastication the food becomes mixed with the saliva which is secreted into the mouth from three distinct pairs of glands. These glands, termed the parotid, submaxillary and sublingual, do not secrete exactly the same kind of fluid. It is not difficult to collect the fluid secreted in the case of the parotid and submaxillary glands by the insertion of cannulae into their excretory ducts, and by other means in the case of the sublingual gland, it being found that the saliva varies in properties according to the gland from which it is derived. Parotid saliva, for example, contains no mucin, and is thin and watery, and in most animals is particularly rich in ptyalin as compared with that of the submaxillary and sublingual glands. The secretion from these latter glands is rich in mucin and hence is somewhat viscid and stringy.

The saliva of the mouth then is a mixed fluid derived from the three pairs of salivary glands, and from some of the smaller glands of the mucous membrane of the mouth. As obtained from the mouth it is frothy, opalescent, turbid and somewhat viscid. The specific gravity varies from 1.001 to 1.008 and the reaction is normally alkaline, although it may be neutral to even acid as a result of fermentative processes in the mouth, or in certain disorders of the alimentary canal. The relative amounts of the constituents, 99.5 per cent of which is water, will depend upon the relative degree of activity of the different glands, and this in turn will be determined by the character of the food. When the food is dry, an increased parotid secretion is noticed, the glands apparently adapting their actions to the character of the food. The secretion of saliva is a complex act and involves the co-operation of gland

cells, blood vessels, efferent and afferent nerve fibers contained in certain of the cranial nerves, and a central mechanism in the brain which is located in the medulla oblongata. Normally the flow of saliva is the result of reflex nervous stimulation, the sensory impulses being carried to the center in the medulla and thence transmitted through the Efferent nerves to the glands. Various observers, including Pawlow, have found that the three large salivary glands respond normally to different stimuli. In the dog it was found that when fistulas were made of the ducts that the submaxillary responded readily to a number of stimuli, such as the sight of food, chewing of meats, acids, etc., while the parotid seemed to react only when dry food, such as bread, dry powdered meat, etc., were placed in the mouth. Psychical influences, such as the sight and smell, or even thought of savory food, and the feeling of nausea preceding vomiting are usually accompanied by an increased salivary secretion due to stimulation of the salivary secretory center by nervous impulses descending from the higher nerve centers. This center may be inhibited as well as stimulated as the well-known dryness of the mouth accompanying fear, embarrassment or anxiety in the human will show, or the dry hot mouth which accompanies digestive disorders in animals.

Experimental evidence shows the following results regarding salivary secretion in herbivora. During mastication the parotid on the side upon which mastication is occurring, is actively secreting, while the other is practically resting. When mastication changes over to the other side the reverse takes places. When the horse stops eating the parotids cease secreting, but this is not true in ruminants where they continue to act not only during rumination but during abstinence. The secretion from the submaxillary and sublingual glands is constant from both sides in all herbivora, no matter what the direction of mastication. The submaxillary glands cease to secrete during rumination, the sublinguals are not affected, and the parotid secretion is unilateral to the extent that more is obtained from the side on which the jaws are active. The total secretion in herbivora has been found to depend upon the food; only half the daily average is secreted if green food is used entirely, and only one-third if roots form the diet. Oats increase the amount secreted. Hay absorbs four times its weight of saliva, oats a little more than their weight, and green fodder half its weight. Various drugs such as arecoline, pilocarpine, atropine, etc., as the practitioner well knows, have a marked effect upon the salivary glands, usually through their influence upon the nervous control.

The saliva fulfills two functions, a mechanical and chemical

one. The first function is to moisten the food to assist mastication and to lubricate it to facilitate deglutition. The presence of the mucin is of special value for the latter process, and in some animals such as the carnivora, where no digestive ferment is present, this represents its whole function. Claude Bernard sites the following experiment in confirmation of the above point; an esophageal fistula was made in the neck of a horse and the animal fed wet oats. In one minute 55 grams of oats came out of the opening. After ligaturing the duct to shut off the salivary secretion, the process was repeated and only 14.4 grams came through in one minute.

In man and most herbivora the saliva exerts a marked digestive effect on one of the foodstuffs, namely starch. Two enzymes are contained in the saliva, ptyalin being the principal one, and a small amount of maltase. Under the influence of ptyalin the starch molecules take up water and split into simpler molecules the end result being maltose and dextrin, which are further acted upon in the intestines and converted to simpler form still before absorption. The fact that ptyalin will not act in an acid medium being destroyed by .003 per cent of free hydrochloric acid, together with the short time the food remains in the mouth, has raised the question as to what extent the action of ptyalin on starches should be emphasized, inasmuch as the acid stomach contents would supposedly destroy the ptyalin of the swallowed saliva. Recent increases in the knowledge regarding stomach digestion show that starchy foods mixed with weak alkali remain alkaline for considerable time in the stomach. The acid secreted unites first with the protein, and it is a well-known fact that the stomach contents are not rapidly churned, hence it is some time before the stomach contents are completely acid following a meal. Hence, starch conversion in the stomach under the influence of ptyalin is an important process, and would appear doubly so in herbivora, especially ruminants, where no acid is secreted into the rumen to which the majority of the food passes upon being first swallowed, whatever acidity present here being slowly developed by fermentative processes.

Deglutition

Under deglutition are included all those processes by which the bolus of food is propelled from the mouth to the stomach. The esophagus of the horse differs considerably from that of the other herbivorous animals. For the greater part of its length, or as far as the base of the heart, the muscular coat is of the striated variety, at this point however the muscular coat changes to the unstriated type and becomes much thicker and firmer, while the lumen is diminished. This thick terminal end is always constricted and even if cut off close to the

stomach will allow no material to escape from the stomach. This is one reason why the horse vomits with such difficulty, and also explains the difficulty of passing the stomach tube at times. In the ox, sheep and also the dog, the esophagus is composed of striated muscle throughout and the termination is not constricted, but rather is dilated. The walls are thin and distensible, and very bulky material may be swallowed, which would choke a horse if attempted.

The entire act of swallowing is essentially a reflex act. The wave of contraction that passes over the esophagus and forces the bolus downward, or even upward in ruminants, is due to reflex nervous stimulation, and is not a simple conduction of a contraction wave over the muscle. The act of swallowing is commonly divided into three stages corresponding to the mouth, pharynx, and esophagus, and inasmuch as the pharynx is a common passage for the air and food, it is important that the passage through the pharynx be consummated quickly. The reflex process of swallowing is very complicated, many muscles taking part, and for an act of such complexity and such perfect co-ordination it has been assumed that there is a special nerve center, the swallowing or deglutition center, which has been located in the medulla. The initial sensory stimulus which seems to be inaugurated by the presence of a certain amount of food in a definite state of moisture, etc., in the back of the mouth, liberates a series of reflex muscular contractions which take the food through the pharynx and end with a peristaltic wave that progresses in an orderly fashion along the esophagus. It is interesting to note that there is an important interdependence between the functions of respiration and deglutition. If an inspiratory or expiratory movement were going on during the act of deglutition, food might be drawn into the lungs or driven into the nasal cavities. Such an accident is prevented by the fact that every act of swallowing inhibits a respiratory movement due to a reflex through the glossopharyngeal nerve, the cessation which lasts for five or six seconds, being sufficient time for the whole series of acts concerned in deglutition.

Stomach Digestion

For simplicity of stomach structure among the domesticated animals, the dog would seem to occupy one end of the scale, and the ox with its complicated gastric compartments the other. This would in a way appear logical inasmuch as it would seem a simpler matter to convert flesh into flesh than to convert grass, hay, grain, etc., into animal tissue. However, the stomach of the horse is simple as compared with that of the ox, and as long as bulky compartments are provided somewhere in the alimentary canal for the maceration of the

fibrous portion of the food in herbivora, it appears to make little difference whether they come first or last. In the ox cellulose digestion takes place largely in the stomach, and the intestines are comparatively simple, while in the horse the more soluble constituents of the food are extracted first, and the macerative processes occur in the great bulk of intestines provided.

The Stomach Glands and Secretions: In the stomach of the horse the stomach wall is composed of four coats—serous, muscular, submucous and mucous. The lining membrane or mucous coat is clearly divided into two portions. The upper portion appears as a continuation of the esophageal mucous membrane, being white in color, containing only a few mucous glands, and covers the entire left or esophageal extremity of the stomach. It constitutes about one-half the stomach mucous membrane. Just below this and to the right, three zones may be recognized, all of which are soft and velvety to the touch. The middle narrow zone, termed the cardiac zone, is yellowish gray in color and contains short tubular cardiac glands. Below this is a large region, the fundic gland region, which is reddish brown in color, and contains the fundic glands. The mucous membrane in this region is particularly thick and vascular. The remainder of the mucous membrane, termed the pyloric gland region, is reddish gray in color and contains the pyloric glands.

The gastric juice is an admixture of secretions from the various glands found in the stomach mucous membrane. When collected in pure form it is quite clear, strongly acid, and without smell. It contains about 0.3 to 0.6 per cent total solids. The actions of gastric juice are due partly to the acid and partly to the combined action of the acid and enzymes. The true acid of the gastric juice is entirely hydrochloric varying from 0.2 to 0.6 per cent in the various animals. Other acids found in the stomach contents, and particularly lactic acid which predominates in certain stages of digestion in herbivora, are produced by processes of fermentation occurring in the food. The hydrochloric acid in the stomach is secreted by acid secreting cells in the fundic region. The enzymes of the gastric juice are pepsin, rennin and gastric lipase. Pepsin is secreted by the peptic cells found in both the fundic and pyloric gland regions as the zymogen propepsin, which is converted to pepsin by the hydrochloric acid, and acts only in an acid medium chiefly upon the proteins of the food, converting them to simpler bodies, peptones and proteoses, the action on which is completed in the intestines.

The enzyme rennin is likewise in an inactive form and activated by the hydrochloric acid. Its function in adult herbi-

vora is apparently not important, in that its action is confined to milk, coagulating the protein casein and thus facilitating its digestion by the pepsin.

The effect of the gastric juice on fats is the solution of their connective tissue framework so as to set the fat free. Some fat conversions may take place due to the action of the warm dilute acid, and fat ingested in a finely divided form as in milk may be hydrolized by the gastric lipase. Such carbohydrate digestion as occurs in the stomach is due largely to the ptyalin of the swallowed saliva, and to the hydrochloric acid, as there is no special enzyme secreted in the stomach for this purpose. Some cellulose fermentation is said to occur in the stomach of the horse, and undoubtedly does to some slight extent, possibly to the extent of the destruction of the cell wall of certain grains. The destruction of the cell wall of oats and barley which is said to occur rapidly in the stomach of the horse is stated by some observers to be due to a cyto-hydrolytic enzyme pre-existent in the grain itself.

The secretion of gastric juice is under the control of the nervous system, and the secretory fibers are contained in the vagus nerve. In an ordinary meal the secretion first started is due to the sensations of eating, that is, it is a psychical secretion and will take place though the esophagus be severed and the food not allowed to enter the stomach. This psychical reflex secretion is supplemented by a further action arising in the stomach itself. It seems as though some foods contain substances designated as secretogogues that are able to cause secretion of gastric juice when taken into the stomach. The researches of Pawlow and his co-workers seem also to indicate that the quantity and properties of the secretion vary with the character of the food, and other conditions being the same with the amount of food to be digested. Different kinds of food produce secretions varying then not only in quantity but also in their acidity and digestive action.

Stomach Digestion in the Horse: The digestive processes in the stomach of the horse have been worked out largely by experimental feeding and destroying the animals at certain stages of digestion, inasmuch as it has not been possible to establish gastric fistulas on account of the distance of the viscus from the abdominal wall, susceptibility to infection, etc. The average capacity of the horse's stomach when fully distended is three to four gallons, and the organ is under the best physiological conditions for digestion when about two-thirds full, i. e., containing two or two and one-half gallons, and in fact rarely contains more than this amount normally.

The stomach of the horse is rarely empty, unless after a period of starvation. When food is taken by the horse, the

stomach becomes gradually distended until it becomes about two-thirds full, owing to the fact that the food which commences to pass out, which was the food remaining in the stomach from the previous feeding, does not pass out at first as rapidly as the new food enters. When the stomach becomes two-thirds full the most active digestion is occurring, and the amount passing through the pylorus into the duodenum equals that entering at the cardia. This continues until the feed is finished, and then gradually slows up to the extent that it would be many hours before the stomach would be completely emptied. No churning of the contents takes place in the stomach of the horse, and the successive feeds are arranged in strata in the order of their arrival, the first food being in the pylorus and greater curvature, the last in the cardia and lesser curvature. Mixing of the feeds occurs only at the pylorus at the time of passage into the intestine.

There has been considerable discussion as to the proper order of allowing the grain, hay and water to horses, in order to secure best action by the stomach. Experimentation seems to show that the practice of giving water first and feed afterwards is correct, owing to the fact that watering after feeding tends to disturb the normal arrangement of feed in the stomach, and may wash considerable food into the intestines before proper stomach digestion has been completed. As far as the grain and hay are concerned, inasmuch as the food first consumed tends to pass out first, and a certain amount may remain until the next feed, it would seem logical to give the least albuminous food first. While this is apparently the reverse of the successful English practice of water, then oats, followed by hay, yet not entirely so. Their idea seems to be that the hay remaining in the stomach from the previous feed will pass out first, and thus prevent the oats from passing out of the stomach too soon.

Ellenberger states that in order for horses to secure the greatest possible nutriment from their oats, hay should be given first, and then water which will carry some of the hay into the bowel and after a time the oats are given. A portion of the remaining hay will then pass into the bowel while the tendency of the oats will be to remain in the stomach. Ellenberger and Hofmeister state that in the horse after a moderate feed, gastric digestion is at its height in 3 or 4 hours, after a full feed in 6 to 8 hours, and after an immoderate feed is delayed still longer.

Stomach Digestion in Ruminants: The stomach of the ox occupies nearly three-fourths of the abdominal cavity, filling all the left half, except the space occupied by the spleen, and extends considerably over the median plane into the right half.

Of the four divisions, the rumen, reticulum, omasum and abomasum, the first three are regarded as proventriculi, the fourth compartment the abomasum, being the true stomach and is the only compartment in which a true digestive secretion containing the stomach enzymes is formed.

The capacity of the stomach ranges from 30 to 60 gallons, varying with the age and size of the animal. At the age of one and one-half years the four divisions have reached their definite relative capacities, the rumen constituting about 80 per cent, the reticulum, 5 per cent, the omasum 7 to 8 per per cent, and the abomasum 8 to 7 per cent of the stomach mass. An important point which is often overlooked by the practitioner, especially in administration of drugs, is the marked variation of the relative sizes of the stomach compartments up to one and one-half years. In the new born calf the rumen and reticulum together are about half as large as the abomasum; in ten or twelve weeks this ratio is reversed. During this period the omasum appears to be contracted and functionless. At four months the rumen and reticulum together are about four times as large as the omasum and abomasum. At about one and one-half years the omasum and abomasum are about equal in capacity.

The Rumen: The cavity of the rumen is partially divided into sacs by what are termed muscular pillars and which as will be seen play an important part in stomach peristalsis. The mucous membrane is brown in color and studded with papillae which vary in form and size, some being foliate, some filiform, and others club shaped. The esophageal groove which is usually mentioned in connection with the rumen is a semicanal about six inches in length, which begins at the esophageal opening into the rumen and passes downward in the right wall of the reticulum to end at the reticulo-omasal orifice. The wall of the canal is incomplete and thus communicates with the rumen and reticulum, at the upper end with the esophagus, and at the lower end with the omasum.

During feeding it is very probable that the products of the first mastication pass almost entirely to the rumen. Some of the very soluble constituents together with a little fluid pass into the third and fourth compartments. The bulk of fluid passing to the stomach passes to the rumen and reticulum, the overflow passing to the third and fourth compartments. Without fluid the rumen cannot function; when the supply is cut off rumination ceases. During feeding the rumen fills up gradually with coarse ingesta and fluid, and finally becomes full, though not tightly packed. The last food to arrive occupies the anterior extremity of the dorsal sac, but from here it passes through all parts of the rumen, for experimental evi-

dence shows that the mass in the rumen actually rotates; during rumination or just after fluids are swallowed, the movements of the mass are most energetic, the idea seeming to be to secure thorough mixing of the contents with the fluid, and also to throw all parts of the mass toward the esophageal groove to give an opportunity for regurgitation and remastication. The contents of the rumen vary in reaction from alkaline due to the enormous amounts of swallowed saliva, to acidity due to fermentative processes. There is no secretion aside from possibly slight mucus from the walls of the rumen. The essential function of the rumen is to receive and retain food for remastication, to macerate fibrous substances and prepare them for cellulose digestion. Cellulose digestion to the extent of 70 per cent takes place in the rumen, largely due to bacterial fermentation, or according to a few observers under the influence of ferments contained in the food itself. Considerable of the first steps of carbohydrate digestion also take place in the rumen due to swallowed saliva. In young ruminants digestion occurs largely in the abomasum, the other compartments, as we have stated, being rudimentary, developing rapidly however as the animal commences to eat more solid food. The rumen is never completely empty except after prolonged starvation. The rumen communicates with the reticulum and the omasum as well as with the esophagus.

The Reticulum: This second compartment is the most anterior, and communicates freely with the rumen over the fold which separates the two cavities. It communicates with the esophagus through the esophageal groove and with the omasum by means of the reticulo-omasal orifice.

The mucous membrane possesses no secretory activity and is arranged in polyhedral cells resembling honey comb. The contents are fluid and alkaline in reaction. This fluid is probably used in rumination as the reticulum is capable of very energetic contractions which forces the fluid over the ruminoreticular separation, floods the rumen contents and also forces it up to the esophageal groove. It is in the reticulum that the various foreign bodies frequently swallowed by cattle usually lodge, and frequently penetrate through the wall and diaphragm into the pericardium causing traumatic pericarditis. The principal function of the reticulum seems to be to assist in the process of rumination by storing fluid for this purpose, and forcing it to the esophageal groove at the proper time.

The Omasum: The third compartment is peculiar in that it works with its contents above both the openings into and out of it. It communicates with the second and fourth compartments by means of a groove which is formed in its ventral

wall and which forms a direct path to the abomasum from the reticulum for fluids and materials fine enough that they do not need further reduction by the horny laminae of the omasum. The interior arrangement of this organ is peculiar, being composed of a series of various sized leaves covered with horny papillae which project backward toward the abomasum. The organ secretes no digestive fluid nor does it absorb, and the reaction of the contents is neutral. The function of the omasum seems to be to grind and reduce such food as has not been sufficiently reduced for proper action by the abomasum. The chief source of its material is probably directly from the esophagus following remastication, and a large amount of the material passes through the groove in the ventral floor of the organ and is not grasped and further ground by the horny leaves. When rumination ceases from any cause, and thus the chief source of fluid to the omasal contents, the saliva swallowed with remasticated food is cut off, the contained ingesta may become hard and caked. The contents of the omasum are normally dry and for this reason this compartment by some observers is said to be the frequent cause of digestive disorders in cattle. Others deny this, and state that the omasal contents seldom if ever become sufficiently impacted to cause trouble. The nervous control of the omasum has not been thoroughly worked out, but it is said to be independent of the other compartments, inasmuch as it does not seem to respond to the same artificial stimulation that they do.

The Abomasum: The abomasum is the true digestive stomach and is the only compartment secreting gastric juice. The enzymes are the same as those described for the horse, and the digestive processes and end products are also very similar.

The Gases of the Stomach: The stomach gases vary quantitatively and qualitatively with the nature of the food. The composition of the gaseous content of the rumen varies not only with the type of food but with the nature of the fermentative processes. Carbon dioxide is always present in from 50 to 80 per cent of the total, the more easily fermentable the feed the larger the amount. Methane or marsh gas comes next, varying from 16 to 39 per cent of the total. Marsh gas is derived from cellulose decomposition and is found in larger amounts than the carbon dioxide in chronic tympanites, while in acute tympanites carbon dioxide predominates. Sulphuretted hydrogen, nitrogen and oxygen, the latter two derived from the swallowed air, are also present, although the oxygen may be entirely used up during the fermentative processes. The fact that easily fermenting food such as clovers, alfalfa, etc., particularly when they are in the wet state and fine in

texture, produce enormous quantities of gas in a short time, is well known to the practitioner.

Rumination and Vomition: Rumination is a reflex act and can only be brought about by the united action of the diaphragm, stomach walls, and abdominal muscles. A certain distension of the rumen is essential as well as a certain fluidity of the contents. A diverting of the secretion of the parotid glands, which function continuously in ruminants, will in a day or so result in very difficult rumination. There is some difference in opinion as to just how the mass of food for remastication, which weighs about four ounces, is separated from the mass in the rumen, and projected into the esophagus. It is generally considered that during the churning movements of the rumen the material is pressed forward toward the esophagus by the simultaneous contraction of the abdominal muscles and the walls of the rumen and reticulum, with the diaphragm fixed. By these means some of the fluid from the reticulum and semi solid material from the rumen is forced toward the esophageal groove, the bolus being separated from the mass probably by the cardiac end of the esophageal tube, assisted by the lips of the esophageal groove, and by a reversed peristalsis carried to the mouth. The bolus upon being reswallowed does not usually enter the rumen again but passes either to the omasum or abomasum. The fact that one of the earliest signs of abnormal condition in ruminants is a cessation of normal rumination is well recognized.

Vomition among solipeds and ruminants is unusual but may occur. It is extremely difficult for the horse to vomit for a number of reasons, and this inability is the cause of not infrequent rupture of the stomach in this animal. Aside from the thickened termination of the esophagus, the contracted cardia, the fact that the stomach of the horse is not in contact with the abdominal walls, the arrangement of the muscular fibers, etc., all of which would tend to inhibit ready vomition, the fact that there is probably a failure of development of the vomiting center in this animal, or if present is either rudimentary or insusceptible to ordinary impressions is probably the chief cause of the lack of this function. In ruminants also this same fact is probably partially explanatory, because while they have not the mechanical interferences to vomition as does the horse, yet they do not ordinarily vomit.

The Stomach Movements: Stomach peristalsis on the whole is more deliberate and less energetic than intestinal peristalsis. There is no churning movement taking place in the animals with single stomachs but rather waves of contraction which appear at regular intervals and run toward the pylorus, and in this region the peristaltic waves become more

forcible as digestion progresses. These running waves or rings of contraction serve to press the stomach contents against the pylorus. At certain intervals the pyloric sphincter relaxes and the contraction wave squeezes some of the contents into the duodenum with considerable force. The mechanism controlling the relaxation of the pyloric sphincter is obscure. It does not occur at the approach of each contraction wave but at irregular intervals. The consistency of the food probably exerts some influences, however the degrees of acidity on the stomach and duodenal sides of the pylorus probably have the greatest influence upon the conditions of its contraction and relaxation.

The movements of the stomach walls, particularly of the rumen and omasum are very complex as these organs are continually at work. The rumen mixes and revolves its contents contracting energetically about three times per minute. The reticulum and abomasum do not work constantly but have periods of rest. The reticulum is most active during rumination. The movements of the abomasum are simple like those in the single stomach of other animals. The stomach receives nerve fibers from two sources, in the vagi and splanchnics, however it would seem that its orderly movements are merely regulated through these extrinsic fibers, as experimental evidence plainly shows that the primary stimulus to contraction arises within the organ itself, in other words, it is automatic like the heart. Whether this automaticity is a property of the plain muscle of the walls, or depends upon the rich supply of intrinsic nerve ganglia (plexuses of Meissner and Aurbach), cannot be definitely answered at present. The extrinsic nerves not only supply the stomach with efferent fibers, motor and secretory, but also carry afferent fibers from the stomach to the central nervous system. Regarding the efferent action of the extrinsic nerves, experimental results show that the fibers received along the vagus paths are mainly motor, while the fibers coming through the splanchnics are mainly inhibitory. It remains, however, that these extrinsic influences are regulatory rather than bringing about the initial stimulus to contraction which evidently arises within the stomach walls themselves, and is dependent upon certain internal conditions of tension or irritation such as may be brought about by the presence of food in the organ.

The conditions necessary for the development of gastric peristalsis seem to be: (1) a condition of tonicity of the musculature, i. e., a slight degree of contraction whereby the muscle is shortened; (2) intragastric pressure. When these two conditions are mutually adapted the musculature acquires a certain degree of tension whereupon peristalsis arises. An

excess or deficiency of internal pressure as well as a loss of tonicity prevents peristalsis. The effect of an excess of internal pressure in inhibiting peristalsis is well known in conditions of impaction of the rumen, likewise in tympanites in ruminants.

Intestinal Digestion

Very important digestive changes take place in the intestines, and here also the products of digestion are chiefly absorbed. The chyme poured from the stomach into the small intestine is subjected to the combined action of three secretions—the pancreatic juice, the secretion from the intestinal glands (succus entericus) and the bile. These secretions are mixed with the food from the duodenum on, so that their actions proceed simultaneously, however for purposes of description it will be necessary to discuss each separately.

Pancreatic Secretion: The pancreatic secretion is poured into the duodenum by two ducts, in the horse the larger one piercing the duodenal wall about six inches from the pylorus at the duodenal diverticulum, along side of the bile duct from the liver. The secretion is alkaline in reaction, quite clear in color and varies in consistency in different species of animals. According to Delezenne from one and one-half to two liters may be collected from the cow in the course of a day. The important constituents, from a digestive standpoint, are three enzymes or their zymogens, i. e., trypsin, a proteolytic enzyme; pancreatic diastase (amylase), an amylolytic enzyme; and lipase (steapsin), a lipolytic enzyme.

Trypsin is secreted in an inactive or zymogen form termed trypsinogen, which is activated or converted to trypsin upon coming into contract with a substance termed enterokinase, which is contained in the mucous membrane of the small intestine. The physiological value of this relation is probably in the fact that it may serve to protect the living tissues from the powerful digestive action of the proteolytic enzyme trypsin. Both the lipase and amylase of the pancreatic secretion are secreted in active form. The activated trypsin attacks protein in a manner similar to pepsin, its action differing however in several respects. It attacks protein in neutral as well as in slightly acid or markedly alkaline media, and its effect is more rapid and powerful than that of pepsin, the protein molecule being completely broken up. Pepsin and trypsin really act in series, the changes begun by the pepsin being completed by the trypsin.

The diastatic or starch converting enzyme, amylase, of the pancreatic secretion acts in a manner similar to, or identical with, the pytalin of the saliva. The lipolytic or fat digesting enzyme, lipase or steapsin, breaks up neutral fats into glycerin

and the constituent fatty acids, thus preparing them for absorption.

The normal mechanism of secretion of the pancreatic juice is interesting in that the stimulus is derived from two sources and the corresponding secretion varies somewhat in physical characteristics. The secretion seems to consist of two parts: (1) a nervous secretion caused by the secretory fibers furnished by the vagi and splanchnic nerves; (2) a chemical secretion due to the action of a substance secretin. This latter substance is formed by the action of the acid of the gastric juice upon a substance prosecretin present in the duodenal mucous membrane. This substance secretin is carried to the pancreas and stimulates the gland to activity. Secretin is not an enzyme, but is designated by Starling as a "hormone," which is the name applied to a group of substances formed in certain organs of the body, which following their absorption by the blood are carried to other body organs and stimulate functional activity in these second organs.

The Bile: From a physiological standpoint the bile is partly an excretion carrying off certain waste products of metabolism, and partly a digestive secretion playing an important part in the splitting and absorption of fats in the intestines. The exact manner in which bile assists fat digestion is not understood, however it is known to accelerate markedly the action of the lipase of the pancreatic juice in the splitting of fats, and aids materially in the absorption of the products of this hydrolysis. It was formerly thought that bile was an efficient antiseptic and had a restraining influence upon putrefactive processes in the intestines. Bacteriological experiments have shown that the bile itself has very feeble antiseptic influences, and putrefies readily outside the body. However, experiments seem to show that while the bile is not capable of exerting a direct antiseptic action upon the intestinal contents, nevertheless its presence seems to limit in some way the extent of putrefaction.

Although bile is formed more or less continuously by the liver cells, its ejection into the duodenum is intermittent. It enters the duodenum periodically during the time of digestion. During the intervening periods it is prevented from entering the duodenum by the fact that the opening of the common bile duct is closed by a sphincter. According to Bruns, when a meal is taken the ejection of chyme into the duodenum is followed by an ejection of bile. It would seem that each gush of chyme, probably by reflex action, excites a contraction of the gall bladder and ducts, and also an inhibition of the sphincter closing the opening into the intestines.

The Intestinal Glands and Succus Entericus: The glands of the small intestine are of three kinds: (1) The intestinal glands (Lieberkuhn's glands) are found throughout; (2) the duodenal glands (Brunner's glands) are found in the first twenty feet of the bowel; (3) the lymph follicles which are found either scattered or in groups. If scattered the lymph follicles are termed solitary glands, and if in groups, Peyer's patches.

The mucous membrane of the large intestine is thicker and darker in color than that of the small intestine. It contains intestinal glands (Lieberkuhn's glands) which are large and numerous, especially at the apex of the caecum and in the dorsal part of the colon. It has no Brunner's glands or Peyer's patches. Experiments seem to show that the succus entericus secreted by the intestinal glands has little or no digestive action except a weak solvent effect upon starches. Extracts, however, made from the walls of the small intestine have been found to contain four or five different enzymes and to exert a most important influence upon intestinal digestion. It is probable that these enzymes belong to the group of endo enzymes and are not actually secreted into the lumen of the intestine, but exert their activities from or within the intestinal walls.

These enzymes and their actions are as follows:

1. Enterokinase, activates the proteolytic enzyme trypsinogen of the pancreatic juice.
2. Erepsin, acts particularly upon deutero-albumoses and peptones completing their hydrolysis.
3. Inverting enzymes, maltase, invertase and lactase, convert disaccharide sugars to mono saccharides.
4. Nuclease, acts upon nucleic acids.
5. Secretin, not an enzyme but a hormone, being absorbed and carried to the pancreas stimulates flow of pancreatic juice.

Intestinal Digestion in the Horse: The chyme upon passing into the small intestine changes in appearance, the acid albumin being precipitated by the alkaline pancreatic and biliary secretions. The contents vary somewhat in reaction, being slightly acid next to the pylorus, then neutral, the alkalinity increasing as the contents pass backward. A large amount of mucin is present which has probably been derived in the stomach. The small intestines never appear full and often are entirely empty. The contents in the anterior portions of the small intestine are very fluid, while in the posterior portion the fluid material is considerably diminished, a point which denotes very active absorption in the small bowel. No fecal odor is noticed in the small bowel until the ileum is reached.

Water according to experimental evidence, passes very rapidly through the stomach and small intestine of the horse, reaching the large intestine, probably the caecum, in from five to fifteen minutes.

The enormous development of the large intestine in the horse would point to very important digestive changes occurring there. It presents a considerable contrast to the small intestine in that it is always filled with ingesta, the contents are more solid, and remain for a considerable time therein, and no secretions other than the succus entericus are poured into the large intestine.

The first portion of the large intestine, the caecum, is peculiar in that it appears as a sort of a large bag, having a blind end, both the openings into and out of, being close together, the exit being above the inlet. Observers differ as to the chief functions of the caecum, some prefer to believe that its most important function is to hold water which is supplied to the colon as needed to assist macerative processes, also that considerable digestion of the more soluble constituents may take place here together with active absorption. Other observers, including Ellenberger, believe that the caecum supplements the small stomach of the horse, that the entire feed reaches the caecum in from twelve to twenty-four hours and remains there for about twenty-four hours. During this time active cellulose digestion occurs, as high as thirty per cent of the cellulose disappearing before the food passes into the colon. The accumulations of gas frequently noticed in the caecum would tend to support the view that considerable cellulose digestion occurs here. Observers holding to the first view believe that cellulose digestion occurs almost entirely in the colon, and that the material need not nor frequently does not remain in the caecum at all. It is even suggested that the material may pass directly into the colon from the ileum, by an insertion of the ileo-caecal end of the ileum into the colon, thus eliminating any action by the caecum entirely.

The great volume, variations in size in its different portions, and peculiar twists made by the great colon, make it of great physiological interest, and these same facts possibly account for the frequency of digestive disturbances in this bowel. It has been suggested that the object of the marked differences in volume of the great colon may be for the convenience of its accommodation in the abdominal cavity. In describing anatomically it is divided into four portions. In the first and third the ingesta descends, in the second and fourth the ingesta ascends. In the first portion the food is fairly firm, in the second more fluid, at the pelvic flexure invariably liquid, in the third and fourth portions the material becomes slightly firmer, about

the consistency of thick soup with a great deal of gas present which appears as bubbles on the surface. For a short distance in the small colon this condition is maintained, when quite suddenly the contents become solid, a fact which indicates either a very rapid absorption at this point or else great compression.

The large intestine is actively employed in disintegrating cellulose by a process of bacterial decomposition. The cellulose encloses the proteins, carbohydrates and fats, in a sort of framework and until this is broken down the substances cannot be acted upon. Considerable cellulose solution, of course, occurs before the material arrives in the large intestine, but in the case of hay for example, prolonged maceration is necessary to liberate the proteins, carbohydrates, etc. The digestion of this material, after being set free, is carried on by the enzymes, carried back with the contents from the small intestine, along with the bacterial attack which is carried on against protein in particular in the large bowel. While the small intestine is comparatively free from putrefactive processes, in the large intestine putrefaction is noticed throughout, and indeed, is very probably a normal process in the large bowel, being essential to the complete utilization of certain of the food products, particularly protein. As the material moves toward the rectum, it becomes drier and firmer, and formed into balls by the bowel sacs. In the rectum the single balls collect into masses, to be forced out at the next evacuation.

Intestinal Digestion in Ruminants: The intestines of the ox are of extreme length but small in caliber, and although important digestive changes occur in the intestines in these animals, it would appear that their chief function is that of absorption. The biliary and pancreatic secretions are poured into the small intestine and the other enzymes of this bowel are also present. In the small intestine such proteins and carbohydrates as have escaped action, or only been partially converted by the pepsin and ptyalin, together with the fats are rapidly converted into form for absorption, and such proteins, carbohydrates and fats as may have been locked up in this cellulose covering, having been thus set free in the stomach, are ready for immediate conversion when they are thrown into the intestines.

The Intestinal Movements: The musculature of the intestines is arranged in two layers, an outer longitudinal and an inner circular coat, while between these coats and in the submucous coat are present the nerve plexuses of Auerbach and Meissner. In the large intestine narrow bands of pale longitudinal fibers are noticed, particularly where the bowel is sacculated, this sacculation effecting economy of space with

no loss of surface. Two main forms of intestinal movements have been distinguished, the peristaltic and the pendular or rhythmic. The peristaltic movement consists in a constriction of the walls which passes backward from segment to segment, this advancing wave of constriction being preceded by an area of relaxation, the two apparently combining to facilitate the movement onward of the intestinal contents. This wave of relaxation which precedes the wave of constriction is brought about by an inhibition of the normal tonicity of the muscular coats. This two-fold character of the movement is regarded as a reflex which is controlled within the intestinal wall itself through its intrinsic nerve ganglia and their afferent and efferent connections. Normally, the peristaltic movement is in one direction, i. e., away from the stomach. The term "antiperistalsis" is used to describe the same form of movement running in the opposite direction, and although this may be demonstrated under certain conditions artificially, it is said not to occur normally.

The rhymic or pendular movement, first described by Starling and Bayliss, occurs in the small intestine and consists essentially in a series of local constrictions of the intestinal wall, the constrictions occurring rhythmically at the points at which masses of food lie. As a result of these movements the masses of food are segmented into a number of smaller pieces. From time to time the separated pieces are caught by an advancing wave of peristalsis, and moved forward a certain distance where they gather into a new mass and the process is repeated.

The muscular movements of the large intestine are slower than those of the small bowel possibly for the reason that the material must remain a much longer time in the large bowel, i. e., for at least forty-eight hours and possibly for as long as three or four days. There is some evidence to show that the rhythmical movements of the intestines are myogenic in origin, while the more co-ordinated peristaltic movements depend upon the intrinsic nervous mechanism. The intestine is however not dependent for either movement upon its connection with the central nervous system, for like the stomach and the heart it is an automatic organ whose activity is simply regulated through its extrinsic nerves.

Two distinct impulses are conveyed to the intestines by the intestinal nerves, i. e., those for contraction and for inhibition. The motor fibers to the anterior part of the tract are furnished by the vagi, and for a portion of the large intestine by nerves issuing from the sacral portion of the spinal cord. The inhibitory fibers are derived from the sympathetic nervous system.

Although the intestinal movements are automatic and self-regulated, they can be influenced by both chemical and mechanical means. The normal stimulus is probably the presence of ingesta in the canal which brings about a certain tonicity and state of intra intestinal pressure such as was described in discussing the stomach movements. Various gases and acids will stimulate peristaltic movements, violent contractions follow shutting off the blood supply, or again temporarily when the supply is re-established, nervous excitement causes rapid evacuations of the bowels in horses and cattle, ordinary exercise will stimulate peristalsis, and the influence of various drugs, irritants and otherwise, as well as mechanical stimulation by way of massage are well understood. Bulky food is essential for normal peristalsis in herbivora. They cannot subsist upon concentrated food alone. If cellulose is withheld from the diet of rabbits they die of intestinal impaction. It is the cellulose which largely provides the needed stimulus to peristalsis in herbivora. There is little doubt but that the majority of intestinal disorders, the primary causes of which may vary greatly, are eventually due to disordered muscular action in the intestinal walls.

Gases of the Intestine: The largest amount of gas found in the intestinal canal is in the caecum and colon. Very little if any is found in the small intestine. Carbon dioxide and marsh gas form the greatest percentage of the gas content, being produced as a result of fermentative processes and cellulose decomposition. The gas escaping from a cannula following the use of the trochar in either the horse or ox will usually ignite at a distance from the cannula, thus denoting the presence of methane or marsh gas.

Absorption of the Food Products

It was formerly assumed without proof that absorption from the stomach was an important process. Actual experiments show, however, that while absorption of certain products of digestion from the stomach does take place in man, the dog and pig, yet by no means as readily as from the intestines. In omnivora and carnivora the most active seat of absorption is the small intestine, although absorption in the large intestine is also important, particularly upon certain diets. In herbivora, and particularly the horse, absorption from the large intestine must be a very active and important process, inasmuch as such important digestive changes take place in the large intestine of this animal.

There are two general routes to the circulation for absorbed food products. (1) By absorption into the intestinal lymphatic system, thence to the thoracic duct, and into the general circulation. (2) By absorption directly into the venous blood

capillaries of the intestines, thence by way of the portal circulation through the liver and into the general circulation. The greater per cent of digested protein and carbohydrate is absorbed by the blood capillaries and passes through the liver, while the majority of fat is absorbed by the lymphatics and carried by the way of the thoracic duct to the general circulation. Water and salts are absorbed largely directly into the blood capillaries.

The mucous membrane of the small intestine is studded with innumerable structures termed villi whose chief function seems to be that of absorption. They are found only in the small intestine and by means of these structures the absorbing surface of the intestine is increased it is estimated from three to twelve times. In other words, each square millimeter of intestine represents an absorbing surface of three to twelve millimeters. Each villus, of which there are probably 50,000,000 in the small intestine of the horse, has a highly developed lymphatic and capillary blood vessel system, each of which takes up its respective portion of the foodstuffs. No villi are found in the large intestine, there are, however, well developed systems of lymphatics and capillary blood vessels in the walls of the large intestine which absorb with great rapidity. Rectal injections, and hypodermic injections of drugs into the large intestine are absorbed rapidly, and rectal feeding is a common and practical expedient in certain conditions. It is probable that very little if any absorption takes place from the stomach in herbivora, not near so much at least as in those animals which subsist upon the more easily digestible animal foods.

No ordinary food substance is capable of complete absorption, the utilization of vegetable foods being less complete than animal foods. Certain animal proteins may be absorbed to the extent of 97 to 99 per cent while as high as 30 per cent of vegetable protein may be lost in the feces if the vegetable food is in such a form as not to be attacked readily by the digestive secretions. While the seat of water absorption in most animals is in the small intestine, in the horse undoubtedly the majority of the water passes to the large bowel to furnish the fluid for macerative purposes, and is there absorbed.

The Feces and Defecation

The feces consist of the indigestible residue of the foodstuffs, together with unabsorbed digestible substances, water, mucin, bile pigments, inorganic salts, fatty acids, organisms, intestinal secretions, etc. The feces differ widely in amount and composition with the character of the food. The variation in consistency of the feces in the different animals, and in the same animal, are due to the amount of fluid contained. The consistency of the

feces cannot be altered or influenced by the amount of water drunk but rather by the character of the food. Green food produces pultaceous feces in horses, while other food such as hay causes a firm feces. Nervous excitement will often cause a free action of bowels with liquid feces. A knowledge of the normal appearance of the feces and their variations physiologically is of the utmost importance in diagnostic work.

The act of defecation is brought about by a contraction of the rectum, assisted by the abdominal muscles, with the glottis closed. The voluntary factor in defecation consists mainly in the contraction of the abdominal muscles. When these are contracted, and the diaphragm is prevented from moving forward by the closure of the glottis, the increased abdominal pressure is brought to bear upon the pelvic and abdominal viscera, thus assisting in pressing the contents into the rectum. In the horse the contraction of the rectal walls alone is sufficient to expell the contents, as is proven by observation, and the fact that the animal defecates readily while trotting. Two sphincters close the rectum in all animals, an external of voluntary and an internal of involuntary muscle; they are presided over by a center for defecation in the cord.

The dark green material found in the intestines of the foetus and which is evacuated shortly after birth, is a product of liver excretion and is termed meconium. It contains biliary acids, fatty acids, pigments and inorganic salts of various kinds.

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PHYSICAL EXAMINATION OF THE DIGESTIVE TRACT AND INDIGESTION IN CATTLE

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The word "diagnosis" literally means, coming from the Greek, to see through something. Around cattle you have often had the feeling that you would like to have X-Ray spectacles or something of that kind, so that you could see through the animal. So when you diagnose a case you really see through that case. The diagnosis is the principal task before the veterinarian, because unless a diagnosis is made it is impossible for you to proceed rationally toward restoring the health of the animal. Before proper treatment can be given or even a proper prognosis made, it is necessary that a diagnosis shall be made. Take a concrete case. A great many years ago a man came to me and told me that he wanted to sell a horse for dissecting purposes. I asked him how old the horse was and he said seven years. I said, "Why do you want to sell him?" "Well," he said, "the horse has lung fever and my veterinarian who has been treating him for the past two weeks, says that you had better kill him. We cannot cure him." I asked him if the horse could travel the distance from his farm to the University and he said yes, he could travel all right. So the next morning he came in, leading the animal behind his buggy. When I got there the horse was hitched to a hitching rack, and some farmer had fed there some few weeks before and the grain or oats which lay on the ground had grown up to a height of probably a foot or two. I noticed the horse looking at that spear of oats in an appetizing sort of way. He looked as if he would like to eat it. I made a very careful examination of the horse and I could not find anything wrong with him. He seemed weak and very much emaciated, but his pulse, respiration and temperature were normal. Auscultation and percussion of the lungs showed them to be functioning normally. Everything seemed to be O. K. except for the fact that the animal would not or did not eat. We put him in a corner of the building and put a speculum in his mouth, and there between the cheek teeth we found a corn cob which had become wedged there. The upper and lower cheek teeth had crushed it so that they came almost in contact, and that object rested on the teeth and was so firmly fixed that it took almost all one's strength to remove it. When that was taken out of the horse's mouth we invited him to feed on a quart of oats which he took with avidity. This farmer took that corn cob and he put it in an envelope and put the envelope in his

pocket. He said, "When that veterinarian sends in his bill, there will be hell to pay." (Laughter.)

This man was a college graduate, a licensed practitioner, who had practiced in that community for over twenty years. It was not so much due to ignorance in his case as to carelessness. I am sure that if a correct diagnosis had been made in that instance that man's bill would have been paid and his reputation, which he lost in that community because this spread all over the community and got into the papers, and it practically killed him as far as his practice was concerned. The importance of a correct diagnosis cannot be overestimated. Upon it depends the prognosis, the treatment and the preventive measures necessary to eradicate the disease process. While it is possible for some people to enjoy certain success in the practice of veterinary medicine, without making very many correct diagnoses, nevertheless in time the layman will discover his weakness in this regard and ultimately will lose confidence in him. A correct diagnosis is 90 per cent of the battle, which the veterinarian must wage against disease.

Diagnosis is an art, not a science. As with most arts, in some persons it appears as a talent. Poets and drum majors are born and not made. To a certain degree this applies to the diagnostician. However, careful, methodical procedure in the examination of the patient, coupled with broad experience, will make a fair diagnostician out of any one. The greater the care exercised, and the more methodical the procedure, the truer is this fact. Diagnosis may be defined as an art of determining the presence of disease changes in or on the body by the aid of changes in the animal's conditions, or some of its organs, which may be recognized through the physician's senses of sight, feeling, hearing or smelling.

The diagnosis consists in four things.

First, determining the symptoms of the disease. That is the first thing a man has to do in making a diagnosis is to get all of the symptoms. Then after the symptoms are gathered he must determine the diseased organ; what organ or organs are diseased. Then, thirdly, he must determine the character of the disease. Ninety per cent of the acute diseases that you treat in practice are really inflammations, some phase of inflammation. You talk about poll evil, lung fever, or inflammation of the bowels, enteritis as you sometimes call it—they are nearly all some form of inflammation. This must be determined.

Then the last thing is the name of the disease. We usually turn that upside down. We want a name first thing. We want to be able, when the owner says, "Doc, what's the matter with it?" to spit out, even before we have examined the

patient, a name; and very often the name we have given in no way describes the disease process with which we have to deal. Too often the name of the disease is emphasized. When you take up your text books, or a book on the diseases of animals, you look in the index for a name, and then you look in the text of the book and you read up all about that name. The name to you often seems paramount. As a matter of fact that would be all right if we had some sort of automatic device, where all we needed to do was to hoist the horse's tail, and a slip of paper would come out of his rectum with the name of the disease on it, then we would go home and read about it with much satisfaction. But as a matter of fact very often the owner telephones you that he has a sick horse, or he thinks there is something the matter with his kidneys, or with its kidneys, or it has the bots, and when you get there as apt as not to find a case of glanders or shipping fever, or something which is so far removed from the hint that he has given you over the telephone, that his hint becomes simply misleading.

In making a diagnosis, the first thing to do is to determine and group all the symptoms which the animal shows. A symptom is any observable deviation from the normal state or condition. While anatomy and physiology treat of normal conditions and functions, symptomotology treats of abnormal conditions and perverted functions.

Symptoms are of various kinds. Some of them are objective, others subjective; some are local, others are general. Subjective symptoms are not available to the veterinarian, and are much overemphasized by the physician. Sometimes they do not make a correct diagnosis. They are not any farther along in that regard than we are. How often you go into a doctor's office and tell the doctor that you have a headache. He takes down a bottle of colored tablets, labeled Parke, Davis & Company's headache tablets, and says, "Take one every three hours." He doesn't know what is the matter with you. He doesn't often know what is in the tablet, but he puts two and two together, and according to his very simple formula, headache plus tablet, it will cure. And, of course, the most of them recover.

We veterinarians, of course, do not have subjective symptoms to guide us. An old cow does not whisper in our ear where her pain is. We have got to go by the objective symptoms, those which we can gather with our senses of sight, feeling, smell, etc.

Symptoms which are derived directly from a diseased organ or part of the body are called local, and symptoms which indicate a sympathy on the part of the whole organism are spoken

of as general symptoms. For instance, in pneumonia, the symptoms brought out by percussion and auscultation of the thorax are local; the fever, depressed sensorium, etc., are general.

Further, direct and indirect symptoms are spoken of. A direct symptom is one which emanates from the diseased organ or part of the body alone; an indirect symptom would be one which appeared either as an elimination of the diseased organ or from some other organ which is also and usually involved with the organ primarily diseased. For instance, in bronchitis, the characteristic rales heard on auscultation would constitute a direct symptom, while the nasal discharge, cough, etc., would be indirect symptoms.

So that no symptoms be overlooked, a method of procedure should be adopted. Whether the practitioner uses a method of his own or that of some one else, makes no difference, provided the method employed causes a thorough and searching examination of the patient to be made. The following order of procedure is very thorough and inclusive:

First of great importance is what is technically called the anamnesis, or the history of the case, and I think the older we grow in the profession, the more we appreciate the anamnesis or the history of what has preceded our visit.

First, under the history we get any prodromal symptoms which the animal may have shown before our arrival, and the duration of the disease.

Second, functions of organs; appetite, thirst, deglutition, rumination; feces and urine, frequency, amount, condition; cough, lactation.

Third, probable cause of the disease; infection; animal parasites, intoxication, traumatism, refrigeration; over-exertion, railway transportation; improper care, environment, use.

Food and water; condition, quality, amount, composition, temperature; condition of pasture in regard to possible infection or invasion (anthrax); blackleg, etc.

Fourth, previous diseases.

Fifth, sporadic; enzootic or epizootic.

Lastly, under anamnesis, what treatment has been given.

And so, by taking all of these things together, we can often arrive, or sometimes, even at a diagnosis of the disease, but we can tell at least a great deal better about what is to follow when we have gotten a clear history. Sometimes, of course, this history has to be obtained from the reluctant owner or more often from a reluctant attendant who does not want to assume any responsibility. I remember one time a man came to me and told me that he had a horse which was dead, and I

said, "I am not an undertaker." He said, "Well, I want you to see it." This was a driver for a laundry company. We found the horse lying in a stall, in an apparently comatose condition. I will not go into the details of the symptomotology, but we examined it, and I came to the conclusion that the horse was suffering from colic, a form of indigestion, and that this man had given something to the animal. He swore he had not given the animal anything. But I came to the conclusion that he had given it a very large dose of opium in the form of tincture of laudanum, and the first thing we had to do was to give an antidote for opium poisoning. When the horse came out of that he began to show his bellyache. We afterward found a bottle which smelled of whiskey and laudanum, and I went to the drug store a week afterward and the druggist said to me—in the meantime the horse recovered—"How much laudanum would you give a horse?" And I told him. "Well," he said, "there was a fellow came in here the other day and he said he wanted a quart of whiskey with six ounces of laudanum in it to give to a sick horse." And he had given that to that horse. He felt that he had done a wrong thing and he had the responsibility of the loss of the horse resting upon him. He would lose his job with the company, and so on.

So it sometimes takes a good deal of tact, the same kind of tact that a good lawyer uses when he has a reluctant witness under cross-examination. Sometimes in a tactful way you can bring out things in the history that are important.

Then we have next the signalment, the kind of animal, its weight, its race, color and markings, its age, sex and the use to which it has been put.

Then, third, the present condition of the animal.

Here usually we begin with the conformation and condition; its habitus, its behavior, its facial expression, its countenance, attitude and complaints.

Then we take up special matters, the skin. Under this heading, an examination of the hair; next, the color of the skin, the presence of pigments, icterus, paleness, redness and cyanosis.

Under the next heading, moisture, sweat, oiliness, exudates, blood; then we notice whether any odor is present. Then the temperature is examined, its distribution, the temperature of the diseased parts of the skin. The elasticity of the skin is noted. Also whether itchiness is present. I remember a man who came to me and said he had a horse with a very severe blood disease. I asked him why he thought so. He said it was very itchy, so much so that it was impossible for him to harness the animal. An examination of the patient, after I had

gone over everything else and excluded everything else except some local skin disease, developed that the horse had ordinary horse mange, and a microscopal examination of the scrapings showed the presence of the causative organism of that condition. After treatment for the mange was given the symptoms of the blood and nervous disorder which some veterinarians thought he might have, disappeared.

Next to be examined are swellings, such as oedemas of the skin, emphysema, and circumscribed enlargements.

Also, loss of substance is to be examined for, excoriations, ulcers, atrophy and necrosis.

Then eruptions, spots, papules, vesicles, pustules and everything of that kind; all these must be taken into consideration.

Then we examine the lymph glands and vesicles. The glands are examined by inspection and palpation, as to their size, form, surface, consistency, temperature, sensitiveness and mobility.

The vessels are examined by inspection and palpation.

We examine the visible mucous membrane of the eye especially in horses. The mucous membrane of the mouth is oftentimes also examined. We examine the mucous membrane for many reasons. In the first place, we note whether there is any local disturbance there, but also in general diseases we examine to determine really the condition of the blood. We note, for instance, that if the mucous membrane is very pale and white, that there has a great loss of blood taken place, or, on the other hand, that the blood has been disintegrated, and that there may be present the condition of leukemia or anaemia or something of that kind; or if the mucous membrane is of a very dark color, bluish, we know that the blood is highly charged with carbondioxide, which is a very serious condition. If it is yellow, we know that bilirubin is present and that the bile coloring matter has permeated into the blood, and so on.

Then we take the internal temperature. This we measure per rectum.

The respiratory organs. I will not bother with that now, because we will take it up later.

All the circulatory organs are examined, and then we come to the digestive tract.

First, the appetite of the animal is examined, and its thirst; secondly, we make an examination of the digestive tract and the animal's manner of eating and drinking, its prehension of food, its drinking of water, mastication, deglutition.

Third, rumination; when it begins. Number and method of mastication.

Fourth, gullet eructations. In the horse, they are absolutely abnormal, belching and eructations from the gullet, but in the ox they are often physiological. Then, vomiting; the quality, odor and appearance of what the animal vomits. Animals actually rarely vomit from the true stomach. We very frequently have in cattle regurgitation from the paunch, but that is not true vomiting. True vomiting is not always a symptom of rupture of the stomach when it does occur. Many years ago I was taught that whenever a horse vomited his stomach was ruptured. So a neighbor called me out one night while I was in college, and he had a horse lying there on its breast, and its sternum in the stall, and I sat on an inverted water bucket and observed the condition of the animal very carefully. I did not know enough to examine that animal and I had not any idea what was the matter with it, and all of a sudden the animal extended her neck and there gushed out of each nostril a bucketful of stomach contents. I rose also and I said to the owner, "That horse has a rupture of the stomach, just as I knew when I first saw it and the best thing you can do is to lead the animal out and tie it to a tree where you want to bury it. And in the morning, why dig a hole and put the animal into it." Ten days later I saw him plowing corn with that mare and he said to me, "Doc, I expect that mare would be dead, but she had eaten about all the leaves she could reach on a tree and began to chew the bark and I knew that rupture of the stomach was a matter of only a few days and I thought I would get all the work I could out of her before she died." Ten years later she was killed on a railroad crossing. (Laughter.)

But in about two-fifths of the cases where the horse vomits, the stomach is ruptured. But that one symptom of itself must never be taken. If I had stayed there a little longer and examined that animal's pulse, respiration and temperature, or her other symptoms, I would have saved myself the embarrassment which followed that experience.

Then we examine the mouth cavity; this applies to horses as well as cattle. We make inspection and palpation of the cavity. This is very important. In a case in a cow which would not eat and which showed a bloody discharge from the mouth and which was diagnosed as a sarcoma by a good friend of mine—and he is a pretty careful practitioner, too—an examination of the mouth showed that she had swallowed a fishing line wrapped around a piece of cigar box. She had swallowed it as far as the pharynx and the hook had caught there and stayed. She showed symptoms which made me at first suspect that she might have anthrax or hemorrhagic septicemia or some horrible condition of that kind.

In the ox, the dental pad is important to examine, especially where no mouth disease has existed. The mucous membrane should be examined, the lips, the cheeks, teeth, hard palate and tongue.

Under the sub-head of the pharynx, the throat region should be inspected and palpated, the throat should be palpated internally. In horses a very useful instrument is the so-called rhinolaryngoscope, which can be inserted through the nostrils, and through which you can look and get a fair view of the larynx to a certain extent and of the pharynx.

Then comes the examination of the esophagus. We examine that externally by inspection and palpation.

The examination of the abdomen. By inspection we determine the shape and size of the abdomen, we examine it by palpation, we note its tension and so on. I cannot take this all up in detail, so I am just going in order to give you an idea of how great a task it is to make a thorough examination of the patient and to collect all of the symptoms to make a diagnosis from.

And we carry this along from the abdominal organs over to the urinary organs, and the reproductive organs, and so on. And as a matter of fact we have found it expedient to furnish each one of our students with printed blanks, and whenever a patient comes in the student is supposed to have that blank and to go over the patient and to put down with a plus mark everything he finds. If nothing is found he puts down a zero over it. By reading that over we know that he has gone over the animal and he gets into the habit of doing that. I do not mean for an instant that it will be necessary for you in every case to make such a searching and thorough examination.

After the symptoms have been gathered, grouped and analyzed, the next step is to determine what organ is diseased. Unfortunately, and I want to emphasize this, unfortunately, there are very few symptoms which point with certainty to the affection of a definite organ. In other words, pathognomonic symptoms are rare. In determining the diseased organ primarily affected, it is necessary to thoroughly understand the pathology of that organ. As an illustration, suppose that the symptoms we have gathered indicate that a horse is suffering from some lung disease. While this may satisfy the owner, it should not satisfy the practitioner, who must go one step farther and determine what kind of a lung disease he has to deal with. Obviously, a knowledge of the pathology of lung tissue is essential. The direct symptoms obtained by auscultation and percussion will of themselves not suffice. A flat sound under the percussion hammer does not mean that the animal has pneumonia. It simply tells us that the lung or a

portion of it contains an abnormal amount of liquid or solid matter which has disintegrated the air and the gases normally present. By taking the symptoms obtained by percussion in connection with the general symptoms and the history of the case, the pathological lesion in most cases may be determined. That is, we may have a flat sound on percussion, and if we have with it a rapid pulse, high temperature, loss of appetite, constipation, increased respiration, and so on, we can say that that comes from a consolidation of the lungs due to inflammation, and we call that pneumonia.

The recognition of all the organs diseased does not of itself suffice. The organ primarily diseased is obviously the most important. Very often we go astray there. We open a horse's mouth and we get some little mouth lesion, and we assume that that is the sole trouble with it. A better illustration is this. I do not know how often this mistake is made. In communities where they have had veterinarians for a long time it has become customary to have the teeth dressed. The owners drive up to your place of business and they want their horses' teeth sharpened. Very often thoughtlessly you dress that horse's teeth without taking its temperature. You find two or three days later that that horse is being treated for pneumonia by your competitor. What the owner will say to you will not sound pleasant to your ears. So we have made it a rule that under no circumstances will we dress the teeth of any horse without first taking his temperature. We learned that from bitter experience. Sometimes the owner of the horse says that the horse does not eat and is constipated, or the ox for that matter, and a great physic ball is given, and superpurgation results, and your competitor is called in, and he finds that you have been treating the wrong end of the animal, that the trouble was pulmonary, in the lung, and your remedy was applied to the bowels.

If our treatment be directed against the primary disease, and its healing brought about, organs secondarily affected will also regain their normal condition and activity. The converse, however, is not true.

Once the character of the disease is determined, the next step as noted is the name. The value of a diagnosis depends upon whether it is made for a scientific or for a wholly practical purpose. As a rule in practice the diagnosis is made from the latter standpoint. In many cases the diagnosis is merely symptomatic, such as diabetes insipidus, colic, etc. Now there is not any such thing as colic. Colic is just a way of saying that the horse has a pain in its belly. There is no disease of colic in any such sense as that of tuberculosis or strangles or anything of that kind. So a diagnosis of that kind is merely

symptomatic. In other cases it may be purely anatomical, such an inflammation of the bowels, eczema, etc. In many instances neither the cause nor the lesion in the diseased organ are determinable, in which case the diagnosis is purely symptomatic, like epilepsy, for instance. An ideal diagnosis would be one which would include both the anatomical and the etiological factors (skin glanders, acarus mange, verminous bronchitis).

"Skin glanders," that is a good diagnosis, both as to the character of the disease, what the disease is and where it is. The difficulty in diagnosing internal diseases will obviously depend upon the character of the disease. In some cases the diagnosis is easy, and in others extremely difficult. In diseases like epilepsy, parturient paresis or tetanus, from the anamnesis alone a definite diagnosis can often be made. In other instances even a most searching examination of the patient will fail to determine even the organ diseased. In these instances only a probable diagnosis can be made (internal tumors, abscesses, degenerations in organs rarely diseased, as in the pancreas, etc.)

A diagnosis, therefore, may be a definite, probable or sometimes only a possible one. So much for diagnosis in general.

Now, as applying this actually, I am not going over this long list, so I have just picked out one or two items which we can emphasize, for the time is short, and the rest you can get some other time.

Now, the appetite; the appetite for food. Appetite and a desire to eat (hunger feeling) are not synonymous terms. An animal may desire food and still not eat, due to pain, poor gastric secretion or deficient peristalsis.

To reckon with appetite we should know something of dietary, have experience with animals, and a knowledge of the physiological conditions which affect appetite. Physiologically, the kind of food, sudden changes in food, the conformation of the animal, exercise, temperament, time of year, psychic influences, change of location and idiosyncrasy play important parts.

I owned a horse once which would not eat bran nor chopped food. That was just an idiosyncrasy. I would rather eat copperheads than cucumbers. That is another idiosyncrasy. Those are physiological things which govern the appetite, and they are to be remembered. I traveled fifty miles to see a very valuable trotting horse which had refused to eat its oats. When I got there I found a dead rat in the bottom of the manger. After removing the dead rat, still the horse would not eat the oats until we provided him with a clean, sweet box, and then

he would eat his oats. I don't blame the horse in that case as much as I would the groom. And that was hardly an idiosyncrasy, except that this animal was careful about the hotel that he patronized.

The appetite can be affected both quantitatively and qualitatively. When the appetite is diminished we call it anorexia. In anorexia the animal does not eat up all of its food ration, or does so abnormally slowly. They may begin to eat with avidity, but toward the end of the meal they eat more slowly, the masticatory movements becoming slower and slower. Sick animals may refuse one kind of food, but eat another. Some feeds may be entirely consumed and others only partially, or remain untouched. Sometimes the appetite seems to be for dry and at other times for moist feeds. The appetite may be completely arrested, in which case no food at all is taken.

Anorexia in disease speaks either for a severe internal disease or a disease of the digestive tract. In the first instance the patient may be suffering from an acute febrile disease, or something may be wrong with the teeth, mucous membrane of the mouth, pharynx, esophagus or stomach. If the patient is fed entirely on dry feed and not enough water allowed, the appetite for this feed will be lessened. Occasionally the peculiarity of the appetite may indicate a certain disease. A marked desire to eat, which is soon appeased, is noted in painful conditions of or where some mechanical obstruction is present in the mouth, pharynx or esophagus, causing the act of eating or swallowing to be interfered with. Later in these instances the animal may show no desire to eat. A capricious appetite most often accompanies chronic disease, especially of the digestive tract.

From the standpoint of prognosis, a prolonged anorexia is an unfavorable sign. However, there are exceptions to this rule. In some animals which lack vigor and are easily excited, food may be refused which would be eaten by other animals suffering from the same disease. In acute feverish diseases the character of the infection and the degree of intoxication are factors governing the appetite. Not infrequently patients with high fever will show good appetite, as has been noted especially in the early stages of strangles, infectious pneumonia of the horse, Texas fever in cattle, etc. Many horses suffering from pneumonia will eat well during the entire course of the disease. Here we assume, of course, that the stomach is still intact.

The appetite may be abnormally increased. In diseases where the consumption of food material in the body is unusual and the function of the digestive tract unimpaired. Therefore in diabetes mellitus, which is a very rare disease in ani-

mals, but common in man, where parasites exist in the blood and in the bowels or the intestine, the appetite of the patient may be extremely good. It may eat so much that it makes it poor to carry it around, to use a common expression. So in spite of this good appetite the animal does not thrive. The same is true to a certain extent in some advanced cases of tuberculosis, provided the digestive tract is in good condition, and in cases of pernicious anemia. As a rule the polyphagia appears only in the advanced stages of these diseases.

Then comes the matter of thirst, which we have not time to touch upon.

The perverted appetite. Perversion of the appetite is shown by the desire of the animal to eat unnatural or even disgusting substances, which would not be eaten by healthy animals. Healthy animals in play or from habit sometimes eat unnatural things. Cattle have a penchant for consuming clothing, old shoes or bones. I remember a few years ago a friend of mine had charge of the meat inspection in Kansas City and there was an Israelite who made a practice of buying lumpy-jawed cattle, cattle suffering from lumpy-jaw or actinomycosis. These cattle he sold subject to inspection. As a rule, in that disease, if the animal's general condition is good they simply destroy the head, and the carcass goes through as first-class meat. So as a general proposition this Jew was making a lot of money, and he bought a farm out in Kansas from some veterinarian. One day a very fine bull, two years old, a white-faced bull, came there with a lumpy jaw, and he went to my friend and he wanted to know how much he would take to cure that bull. He said, "I will take fifty dollars." "Well," he said, "all right, but you have got to put up the money in the bank so that I can get it out." He said, "Won't you trust me?" He said, "No, not in a case of this kind." So he put up the money, and he reached in the mouth of the bull and pulled out the proximal end of a humerus or the upper arm bone of an ox, which the bull in play had gotten into his mouth, in between the cheek teeth, and which made a beautiful actinomycosis looking tumor, which it cost the owner fifty dollars to have removed.

Cattle like to eat clothing and things of that kind. In all of these instances, however, the appetite for other food is retained, and the general condition of the animal is good, provided, of course, the foreign bodies they swallow do not injure the digestive tract.

A perverted appetite which is transient accompanies certain digestive diseases. During the stage of convalescence from severe general diseases, and not infrequently from lack of salt or lime in the food and water does this condition occur. In acute and chronic catarrhs of the stomach the patients often

crave alkaline, bitter and earthy material, because there is a hyperacidity of the stomach present. They, therefore, eat straw or hay which is coated with urine or manure, or lick the walls of the stable which have been whitewashed, or sometimes eat cold objects like stone or pieces of iron. In these cases the cause is partly some disturbance in the innervation of the digestive canal, partly an abnormal condition of the blood which may lack salt or lime, and sometimes this may come from a hyperacidity of the stomach. Perversion in appetite is often expressed by sows eating their pigs and cattle eating the afterbirth. Horses have been known to eat raw flesh, mice, rats or even sparrows. We had in Columbus a most interesting instance of this kind. A horse which was an old rat-eater became afflicted with what seemed to be at first glance a case of chronic tetanus. The animal did not recover, but seemed to be slowly getting worse. The horse was an old one, which had seen many years of service hitched to a hardware man's wagon, and on post mortem we found some little peculiar pin-points scattered throughout the muscles. A microscopical examination of these muscles showed that the horse was suffering from trichinosis, a common disease in pigs and uncommon in rats, and that was one of the two cases that have been recorded, of trichinosis in the horse.

The perverted appetite noted in rabies is a matter of common knowledge. You have seen cases of rabies where the animal would refuse natural food, but would turn around and lick up its own urine and feces.

A permanent perversion of appetite is observed in pica of cattle and wool-eating of sheep, and feather-plucking in chickens. I do not know how many sheep you have in this state, but our state used to be quite a sheep state and I have seen a bunch of sheep pick out one from the flock and they would browse all the wool off of that sheep, and when they would eat up all of his wool they would pick out another and that was pretty hard on the wool business. Those are instances of perversion. Both of those conditions seem to arise where the food contains too little salt, phosphorous or lime. It is probable, however, that certain toxins develop at times in the food which when ingested disturb metabolism and arrest normal blood and bone formation. Perverted appetite is frequently the forerunner of osteomalacia and rachitis in cattle and swine. Therefore, in all cases of continued perversion cattle should be examined for these diseases. However, it may occur independently.

Closely allied to appetite is rumination, about which Dr. Bergman told you so much this morning that I can only be brief about it. When a ruminating animal chews its cud, it in

reality is merely "eating its dinner." The cow evidently in her earlier state was a timid creature, surrounded by all sorts of wild carnivorous beasts, and in all probability she stole out from her lair during the night season and with her tongue and her peculiar incisors just pulled the grass in and made a few very superficial and rapid masticatory movements, swallowed it as fast as she could, where it went into her rumen. The coarse, unmasticated food which has been hastily swallowed and allowed to accumulate in the rumen is vomited up, so to speak, into the mouth, to be carefully re-chewed, mixed with saliva and re-swallowed. Most of it passes to the omasum and abomasum, provided it has been thoroughly masticated and crushed and ground and mixed with saliva.

We made some experiments to see where the water went when given to cattle. We took some cows that were condemned on the tuberculosis test and let them go without water for a couple of days and then permitted them to drink, and in some cases we drenched them along the lines of least resistance with water colored with lamp soot. That was better than anything we could find. We had to color the water. Then we pithed these cows and made post mortems as rapidly as we could, to find where this black was distributed. We found it everywhere. There was a good deal in the reticulum, over the dam or partition which partially separates the rumen and the reticulum; we found a good deal of it in the anterior part of the rumen. There was little in the omasum except at the opening below; the larger quantity of it was in the abomasum. Of course we did not make this experiment quantitatively. I could not tell you what per cent went here and what per cent went there.

A friend of mine, a veterinarian, who killed sheep and rough-dressed lambs for the New York market, made similar experiments with ink, I think he used, with similar results. So in all probability where it goes depends upon a good many factors, the kind of food taken, the consistency of the food, the avidity and vigor with which it is swallowed and so on.

The act of rumination in the ox is extremely important, not only for the functioning of the fore-stomachs, but for the whole animal organism. When it is interrupted, gastric digestion ceases and a marked drying of the contents of the omasum follows. I did not say f-o-u-r stomachs. I said the f-o-r-e stomachs. As you were told this morning, the stomach of the ox is complex. It has four separate departments. Three of these departments are in a sense simply dilatations of the gullet and the abomasum or so-called true stomach is the only digestive stomach. The act of rumination, of course, is to reach the food which has accumulated in the lunch box as we some-

times speak of the rumen. It is not fair to put it this way, because it is not strictly true, but if you can stretch your imagination a little bit, but not too far; when the cow is out eating grass hurriedly, as she usually is, in the manner that I have described to you, she has had her dinner in the sense that your mother or your wife is eating her dinner when she goes out into the garden and gathers sweet corn, potatoes and things of that kind and puts them in her apron. That is not strictly true, because some food when taken by the ox is not regurgitated. All of it is not regurgitated, but nevertheless rumination is very important. Probably you noticed this morning (illustrating on the blackboard). Here the esophagus comes and here is the opening of the gullet, and here is the so-called esophageal groove which is somewhat spiral. Here (indicating) is where the food enters, and here (indicating) is where it leaves. So all this accumulation of food in the back part of the rumen has to pass out here (indicating). After it has passed out through this opening and has been re-chewed and mixed with saliva, it is again swallowed. When the act of rumination is interrupted, gastric digestion ceases, and a marked drying of the contents of the omasum follows.

Importance of digestive disturbances in rumination.

As a general proposition, the disturbance in rumination bears a direct relationship to the intensity of the disease. In disease of the fore-stomachs which interfere with their movements, rumination is suppressed. Sometimes the causes of suppressed rumination are purely mechanical ones, such as adhesions, heavy and blunt objects such as sand, more rarely, enlarged abdominal organs, like the pregnant uterus, or still more rarely a prolapse of the reticulum into the thoracic cavity. I had one case where through a rent in the diaphram the reticulum entered the thoracic cavity. In practice we find the movements of the fore-stomach arrested most commonly from an atony of the musculature. This atony, as was explained by Dr. Bergman, is a lack of tone or tenseness in muscular contraction.

When I was a boy in school we had a drill and one of the movements we called "Inspection Arms." The cadet was supposed to toss the gun up into his left hand, and hold it at the height of his chin. That gun weighed from seven to nine pounds. We used to tell the rookies that the University record for holding a gun was twenty-six minutes, and that the faculty would give a handsome silver cup to any one who would beat that record, and those poor devils would hold that gun as long as they could. One fellow I think held it nine or ten minutes, and the muscles then would relax, and the arm would

become limp, and the gun would light on his toe. That was the object, of course, in the performance.

You all possibly know in human medicine how difficult it is to set a dislocated shoulder joint. We had as one of the professors a man who used to be a football player. He only weighed 200 pounds and we had a rather effiminate student who rode on his bicycle on the paths, and he liked to bump into the professors. He bumped into this man one day and this man did not notice that anything hit him, but the student was precipitated over a wall and over a precipice about ten or fifteen feet high. And he came up to the veterinary hospital, because that was the nearest place for first aid, with his arm out of joint. And there happened to be our professor of physiology, a physician, in the building at the time, and he looked at him and he said, "I want a couple of good strong men." And he sat this patient in a chair and one fellow grabbed him around the waist this way (indicating) and the other one took hold of the end of his arm and pulled firmly and steadily, and the doctor said, "Now, don't relax for a minute. If you get tired tell me and I will have some one take your place." Pretty soon the muscles began to relax, and the doctor put one of his fingers under the humerus, and with a quick movement like that (indicating) it snapped back into place.

You have noticed another thing. Suppose a calf should swallow an apple and the apple become lodged in the esophagus, and as soon as it is dislodged it goes down into the stomach. That is due to the fact that the muscles make desperate efforts to dislodge that apple. They become fatigued and dilated so that as a matter of fact the apple lies in a sort of pocket like that (illustrating). And when you drive it out of the pocket it goes rapidly on its way to the stomach, or up into the throat, depending upon which direction you have pushed it. Now, we have something of the same kind in the stomach of the ox. If it becomes abnormally distended with food there must be an effort for a time for the muscular contractions to overcome this. However, these do not often suffice and as a result they relax. You have a general relaxation of the whole thing, a condition which we now recognize and speak of as atony of the rumen. If the feeding of such food be continued paralysis of the fore-stomachs and usually of the rumen results.

Following severe internal diseases an inflammation of the stomach walls may occur and this leads to paralysis. One of the symptoms of inflammation, as some of you remember, is lost function. If you have an inflammation of the elbow you have a partially paralyzed elbow. This applies just as well

to the stomach. In other cases the movements of the rumen are mechanically interfered with by an abnormal amount of food, which may be good food, however, and the condition results that we call overloading the stomach or paunch.

Then commonly we have in cattle, due to this peculiar way in which they prehend their food, you may find the rumen containing shingle nails, and if the animal is hungry she will eat your watch or your razor or your watch chain, or anything she can get hold of, because, as explained, the act of mastication during the first swallowing is very limited.

Now, we have a great deal of trouble arising from these foreign bodies which get into the stomach. They sometimes go farther than the stomach. I remember we had a cow who had an acute rheumatism. That cow walked stiff. She groaned when she lay down; she did not eat well. We put her on salicylate of soda, which did not do her any good, and then somebody suggested this and that and the other and we tried it, and so on, and finally the head of the animal husbandry department gave us the cow for experimental purposes and I thought it would be interesting to do a rumenotomy. So we gave her an ounce and a half of chloral, tied her to the side of a wall, and we opened through the left flank into the rumen. I do not need to describe the operation, which is a simple one. And we bailed out one-third or one-half of what was in there. Our anatomist wanted to take a lantern and go in there and see what the inside of a live cow's stomach looked like. So we enlarged the opening until he could get an electric light in there, and he went in and inspected. Then I palpated and I found over the right, toward the liver, that the reticulum seemed to be adherent. So I broke down the adhesions. That seemed to cause some pain in spite of the chloral. I picked out a piece of tin, a penny and a few screws and bolts. You can always tell how thrifty the farmer is by examining the reticulum of his cows. While I was palpating there we found the head of a nail, and I pulled on it, and low and behold, it was penetrating through the wall of the reticulum into the liver. I was not in there, but if the anatomist was right, it was into the liver. And we sewed the old cow up, expecting that she would die. She made a brilliant recovery and all the trouble disappeared. I think it was seven or eight years afterwards she was again turned over to us for experimental purposes, on account of old age and we still found the old scar, and there was no longer any evidence of an abscess in the liver, but cicatrix in the liver where this had been. In fact, we have so much of that that we have a special disease, which if I had the time I would like to tell you something about it in detail, that we call traumatic gastritis, which is more common than

we realize, especially where much pain is felt, or where tumbled-down conditions prevail upon a dairy farm.

Rumination may be suppressed in febrile diseases, inflammation of the bowels, in the course of any general disease, painful diseases, cachetic conditions and in many brain diseases. As a matter of fact, you will find rumination suppressed in the ox about where you would find the appetite suppressed in the horse. That is not an accurate comparison, but in a sense it is accurate because for the reasons given, all the food that the animal eats is probably not ruminated. We had the idea that if the animal never was fed roughage, that is an ox had never been fed roughage, it would never ruminate. We saw one case where rumination did not occur until the animal was twelve or thirteen months old. It received absolutely no roughage whatsoever, and no one ever saw that calf ruminate except once. It got after some straw. The calf remained stunted and bony and unthrifty. I do not say it did not ruminate, but no one ever saw it. It died of an intercurrent disease, hemorrhagic septicemia, which killed it, with three or four other cattle in the same barn. On the post-mortem examination we found the rumen about two-thirds filled with the kind of food which had been given it, very largely mash bran, cottonseed and so on. Our agriculturist had fixed up for it a balanced ration, leaving out roughage.

Then as noted this morning, rumination has been known —we used to think the animal could not ruminate unless it had an esophageal groove. Well, a certain Frenchman sewed that up and the animal ruminated just as well. And as you learned this morning, an animal with the reticulum amputated can ruminate. So while we do not understand rumination at the present time, in ruminating animals it will be found when we do understand it, to be a very simple process. I always was interested to know whether or not an ox can ruminate standing on its hind legs, and I made the acquaintance of a circus performer who had two trained bulls, that he had taught to stand on their hind legs or at least with their front feet up on high tubs. He told me he never saw them ruminate in that position, but, of course, that would not show that it cannot be done.

Now, just a few words about the stomach of the ox. The four compartments of the stomach, the rumen, reticulum, omasum and abomasum will be considered separately. The rumen is examined by inspection, palpation, percussion and auscultation, only to a limited extent, however, by the last two methods.

Change in abdominal circumference. Physiologically, the abdominal circumference depends upon the amount of food

contained in the rumen. When the rumen contains little food, the hollow of the left flank is sunken, and the region of the lower left flank is distended no farther than the right, which gives to the abdomen the shape of a pear. After taking food the hollow of the left flank becomes flat to convex, and the left half of the abdomen has a greater circumference than the right.

Pathologically the increase in circumference may be due to the accumulation of gas, solids, or, more rarely, liquids. When gas, the condition is called bloat or tympany. In this case the upper part of the rumen region becomes greatly distended and the shape of the left half of the abdomen approaches that of an apple. Obviously, if what distends the rumen is solid or liquid matter, it will give the abdomen more of a pear shape, but if it is gas it will give it more of an apple shape like that (indicating), the gas being lighter than the liquid or the solid. The abdominal walls fell elastic, distended, and if percussed emit a hyper-resonant tone. This condition can develop rapidly after eating food which readily ferments. It may also come about from stoppage of the esophagus, or, more seldom, from overloading of the rumen with food. An acute filling of the rumen with gas is commonly spoken of as tympanites. The condition may be sometimes chronic. This form occurs periodically in severe cases, appearing regularly after each heavy meal; or, more rarely, it may be continuous. If chronic bloat exists for a long time, pressure of the distended rumen upon the left transverse processes of the lumbar vertebrae may lead to their rotating on their long axis; that is, the spinal column may be caused to rotate somewhat in that region. This finding may sometimes be of importance. I mean that if we see the transverse processes affected by the bloating of the rumen, so that they rotate in that manner (indicating) and assume more or less of that angle (indicating). Chronic bloat may exist notwithstanding the normal condition of the food taken, and be due to the fact that belching is prevented through partial closure of the esophagus from compression above by tubercularly enlarged lymph glands. We have found this to be the most common cause of habitual bloat in cattle. Therefore, whenever an animal in the herd is found to be an habitual bloater, that is she bloats up when none of the others do, on ordinary feeding for instance, this animal should always be subjected to the tuberculin test. Otherwise, it may come from chronic disease of the fore-stomach. That is not common. Also to peritonitis or the occlusion of the stomach openings with hair balls, or the pyloric opening with intestinal stones, both of which are rare.

Consistency of the Rumen. Physiologically, the consis-

tency of the rumen varies, depending upon the quality and kind of food contained. Before partaking of food the wall of the hollow of the left flank feels soft and relaxed. By palpation below the hollow a doughy mass may be felt, which retains finger imprints only momentarily. Immediately after feeding, and when the rumen is filled, the finger imprints are retained normally from five to fifteen seconds, to disappear with the next contraction of the rumen. As we will see later, and as you were told this morning, the rumen is undergoing constant movement. There are about, as can be counted by feeling with the clenched fist in the hollow of the left flank, about every thirty seconds such a movement occurs. You can easily feel it, and in many cases it can be seen provided the animal is not too fat. If you palpate below the hollow of the flank you will be able to indent with the fingers the contents of the rumen, but the impression made with the finger is soon wiped away. On the other hand in impaction of the rumen they will be retained for several minutes, because in those cases the movements of the paunch are either very feeble or do not exist at all in a clinical sense. During digestion and for about one hour after feeding—sooner if green feed—the consistency in the upper flank region becomes soft and elastic, due to the fact that gases which develop in the interim, collect between the upper rumen wall and the mass of food. Through the rectum, the rumen feels firm, accepts but does not retain finger imprints except for a few seconds.

The consistency of the rumen can be abnormally increased. That is, it can be harder and firmer than the normal. When the rumen is distended with gas the enlargement extends upward and downward in the region of the left flank, and on palpation is elastic. On the other hand, if the rumen contains an over-amount of food its consistency becomes firm, sometimes as hard as stone, and will retain finger imprints for two or three minutes.

In cases of paralysis of the rumen, especially if following feeding with dry, indigestible food, the consistency of the rumen is hard. The same condition may also arise from foreign bodies, in cases of traumatic gastritis.

Then we may consider sensitiveness over the region of the rumen. This occurs as the result of inflammation of the rumen wall, which is seldom primary. It may be due to the ingestion of hot drinks, traumatism or infection with the necrosis bacillus. This sensitiveness is more commonly secondary, however, and follows long continued overloading of the rumen, atony of the stomach, sand accumulations or foreign bodies. In diffuse peritonitis sensitiveness is also noted, but it is present on both sides of the abdomen.

Now, as to movements of the rumen. The wave-like contractions of the rumen may be seen and felt in the hollow of the left flank unless the animal is exceptionally fat. The movements do not occur with great regularity. Therefore, it is advisable to watch or feel for them for a couple of minutes.

Generally speaking, there are two movements per minute. We find a great variation here, but generally speaking, there is a movement about one every thirty seconds, or two per minute. The movements depend largely upon the kind of food. Where quantities of concentrates and little roughage are given, the movements are feeble. They are usually more intensive for about two hours after feeding; from four to six hours after feeding they are very feeble. Rumination seems to have little influence upon the movements of the rumen. Severe exercise tends to lessen the number of movements. Moderate exercise increases them.

The frequency and vigor of the movements of the rumen are influenced by disease. In diseases of the fore-stomachs, such as overloading of the rumen, acute and chronic tympanitis, atony and inflammation of the fore-stomachs, the number of movements is lessened and the intensity decreases. In mild cases, so-called simple indigestion, the number of movements may not be influenced, but usually they are not so vigorous. Where no movements exist, the condition of the patient is usually serious. So in our examination of the stomach we always take into consideration the number and the vigor with which these movements are made.

Then we consider the peristalsis. The periodical movements begin with the protrusion of the hollow of the flank, and this becomes clouded at the acme of protrusion, and then momentarily disappears. It gives to the auditor the impression of approaching from the distance, to which it again retreats. It is loudest a short time after feeding. The sound is a dull, rolling one, with which is mixed more or less crepitant or even piping tones.

The rumen sounds vary, as do the paunch movements, with the kind of food and the fullness of the rumen. After dry feeding they are usually louder and sharper than when grain food has been given. About two hours after feeding they are less intensive and softer than immediately after feeding.

Pathologically they are influenced by the same conditions which influence the movements of the rumen.

The reticulum can best be palpated by pressing the abdomen with the clenched fist to the left of and just behind the sternum. As the abdominal wall in this region is rather tense, it is usually advisable to rest the elbow of the examining hand

upon the knee, thus using the muscles of the leg to aid, and for support. If there is any sensitiveness the animal will wince and seek to escape, or not infrequently will kick at the examiner. Sometimes in traumatic gastritis the sensitiveness may extend beyond the region of the sternum to the withers or even to the lumbar region. Sensitiveness in the region of the reticulum is usually the result of traumatic gastritis. However, this symptom taken alone may be misleading, as painful diseases of other abdominal organs or even thoracic organs may sometimes cause sensitiveness over the reticulum.

Finally, the absence of sensitiveness in this region does not exclude traumatic gastritis. In this particular cow that I told you about, with the nail run into the liver, there was absolutely no sensitiveness over the region of the rumen.

The omasum occupies the seventh to ninth intercostal spaces between the posterior border of the lung and the costal cartilages; it is separated from the rib wall only by the omentum and diaphram, and is therefore available for clinical examination. Normally the omasum region is not sensitive. Therefore, when pressure produces sensitiveness over the omasum an inflammation of that organ exists.

We must differentiate between a real inflammation and an apparent inflammation. In any condition of the stomach of the ox, where the animal has not eaten for a few days the contents of the omasum become dry and hard. When the stomach is opened its contents will be found not only hard but covered with a coating of epithelium. That is not a pathological finding. A great many assume that there is a sort of diphtheritic inflammation of the stomach, existing, when absolutely nothing is wrong with the stomach except that it has not been busy for a few days, and it has not received an adequate amount of moisture, and has not been moving as it should move, and the epithelium has begun to cover things. Occasionally we do find an inflammation of this organ, and even necrosis. In this case it was found filled with dry, hard food masses and the walls were necrotic.

The true stomach of the bovine lies immediately against the abdominal wall, and is therefore accessible for palpation and auscultation. However, on account of the rigidity of the abdominal wall overlying it, palpation which is made on the right side under the costal arches, can bring out only a sensitiveness. Sensitiveness over the region of the abomasum is found in inflammation, acute dilatation, occasionally in gastric ulcer and very rarely in torsion of the abomasum.

You will see from this the complexity of the situation. I might say in closing that I have not gotten over one-tenth of what I thought I would get over. It was a good deal like

Artemis Ward's lecture on Africa. He talked two hours and dismissed his audience, and they never got to Africa.

But the stomach of the ox is a very complex and a very highly developed organ. Most of the digestive troubles of the ox, therefore, are found to lie in this organ. As a matter of fact, when you are called out to see a sick ox, or a sick bovine, you can expect to find either some thing wrong with the digestive tract, principally the stomach, the reproductive organs or the udder. You will not treat diseases of the eyes very much and you are not finding very many spavins.

But we are interested here, of course, principally in the digestive end of it and that is confined very largely to the stomach. I do not like to put it in figures, but I think it would be safe to say that of diseases of the digestive tract of the ox, fully 85 per cent are found in the stomach, and most of those are in the rumen. The reticulum gives some, but some of them are in the rumen. That may vary in different localities, due to different practices in feeding, etc., but generally speaking it will hold good.

We have in the dissecting room a specimen that I would like to have you see, which will give you an idea in a concrete way of the topography of the stomach of the ox, its relationship to the thoracic organs, especially the heart. It will help to emphasize what you have heard in regard to the physiology, and what I started to tell you about in regard to some of the clinical features.

ROARING IN HORSES

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I have been asked to fill in a vacancy on the program and I will do the best I can. It is something I have not been thinking about except during the last few days, but it is something with which I am rather familiar, this subject about which I am to speak, and that is roaring in horses, its causes, symptoms and treatment and the results, and I am going to run over somewhat hastily some of those things.

Of course, the horse is not the only animal that roars, but among our domestic animals it is the animal which most often has this trouble with inspiration. The sound is not necessarily a roar. We speak of the roaring of a cannon or a cyclone, or something of that kind. That does not describe the noise at all, because the ordinary sound that the so-called roarer makes is a high-pitch sound. That is a high-pitch sound, when the air is drawn in, as if the air was drawn rapidly and swiftly through a narrow crack. It is a whistling noise. "Whistling" and "high breathing" are more descriptive terms than the word "roaring." But sometimes the horse that at first whistles, later on, when the paralysis becomes more marked upon the affected side, begins to make a roaring sound. Take two strings of a piano, take the base string; it is not very tight, and its vibrations are slow, and therefore, the sound is a low base sound; but you tighten up on that string and the vibrations become progressively more rapid, and when they are very, very rapid you have a high-pitched sound. In the beginning of this trouble, when the paralysis is not complete and the vocal cord is under considerable tension, because the muscle which forms the vocal cord is not completely paralyzed, the sound is high-pitched. Let it go for months or years, and the paralysis becomes more pronounced, the muscle that forms the vocal cord shrinks somewhat, and under tension the vibration becomes much slower, it becomes a lower pitched sound.

So when a horse is brought to you and he makes a high-pitched sound, you may know that this is probably the beginning of the trouble, and that the paralysis is incomplete, or what is called paralysis. But if he makes a sound like a snore, then it is a case of complete, or almost complete, paralysis. So that the difference in the pitch of the sound indicates whether the paralysis is complete or not.

Now, what is it that is paralyzed? The larynx is rather a simple thing when we look at it from inside. If we cut the larynx vertically downward from in front and behind, we find

the cartilages here separated, but here (indicating) is the anterior part of the larynx which is called the Adam's Apple. That is where the two branches of the thyroid cartilage come together, at the Adam's Apple. There (indicating) is one branch, and there (indicating) is the other branch. And here (indicating) is another ring-like cartilage, known as the cricoid, which goes all around the larynx and runs up to the inside of this broad branch of the thyroid. That is the cricoid or ring-like cartilage.

And then, lying up above the cricoid and inside of this branch of the thyroid, there is another cartilage, this loose four-sided plate-like cartilage (indicating same) which is known as the arytenoid.

So we have above and in front of this V-shaped cartilage, with the point of the V forming what we call the Adam's Apple. Behind is the ring-like cartilage which is the cricoid. It is like a seal ring. It is broad. But in front, it is just a narrow ring, only a quarter of an inch wide. That is the cricoid cartilage. Right up above, and lapping over the cricoid, a little bit to the outside, and united to it under my index finger, is this pitcher-like cartilage, and that is just exactly what the name in the Greek means. The Greek word for pitcher is "arytene," and the Greek "eidos" means form or like.

And what happens? Why does a horse roar? Here is the epiglottis, the lid which falls down over the top of the wind pipe when the food crosses it. The food goes into the esophagus, and the animal cannot breathe during the swallowing movement. So that all the use of the epiglottis is to act as a lid to cover over the glottis during the movement of swallowing. If you look down into the larynx from above you will see that it is rather a narrow diamond-shaped opening, the points of the diamond being below and above. Behind the diamond shape is the space between the two arytenoid cartilages, and in front the glottis is bounded by the two vocal cords which come together at the point of the thyroid and run backward, and slightly separate and pass into the arytenoid cartilages which come together behind, so that we have that sort of a diamond-shaped opening between the arytenoids behind and the thyroid in front, forming the glottis, which is covered over by the epiglottis.

When the horse inspires, or takes in air, there are muscles that move the two arytenoids, so that the glottis, instead of being closed, opens up. There are muscles attached to the upper part of the arytenoid cartilage which pull that away from the median line, and thus permit the vocal cords to become separated; but if the muscle on one side is paralyzed, the arytenoid on that side has to remain in position, and the ary-

tenoid on the other side whose muscle or muscles are not paralyzed has to move, so that the opening into the glottis is an asymetrical opening. The bilateral muscles are behind, and on the sides, and when they act they pull the arytenoid out and open up the space in the glottis. The muscles that are involved in separating the arytenoid are two in particular, one muscle which is known as the posterior crico-arytenoideus muscle, because it takes its attachment below to the cricoid cartilage, that broad side of the cricoid, and above it takes its attachment to the arytenoid, so that when that muscle contracts it forces the arytenoid outward and tends to open up the glottis.

In this particular case, while it has been a specimen in Kaiserling for a good while, you can see that the muscle is flattened and its surface is convex. It is an old specimen of a roarer. The animal died from some other disease. Then the larynx was taken out, and it showed the left posterior crico-arytenoideus muscle on that side. You are looking at the back of the larynx, which is pretty nearly atrophied. If that were a fresh cadaver, this would be a pale muscle, shrunken, and this muscle (indicating) would be somewhat brownish red, and not pale.

Then there is another small muscle, the crico-arytenoideus lateralis, which is also involved in the abduction or separation or pulling away of the arytenoid from the median line.

The object of the operation is simply this; we cannot restore power to the atrophied muscle. The lesion was probably of nervous origin, and we cannot by any known method with success restore the motor impulses in this recurrent laryngeal nerve, which innervates the posterior and lateral crico-arytenoideus muscles, and therefore, we have to remove the obstruction.

When air is drawn down rapidly through the glottis, on the paralyzed side, it strikes the vocal cord and pulls that down with it, and it becomes a valve. It distends the ventricle on that side and pulls it down with it and hits the top of the arytenoid and pushes it down with it, like the venous blood running along a vein when it opens up all the valves in the vein.

On expiration that valve is flattened against the side wall and the blood glides over it smoothly. So expiration is noiseless and without difficulty, but inspiration is interfered with because the arytenoid, which cannot be drawn up against the wall, because of the bilateral muscles are paralyzed, is just a loose, flapping piece of gristle or cartilage, and the wind strikes above it and fills out the lateral ventricle and pulls the vocal cord toward the middle. On expiration the cord is pushed

aside, and it does not form any obstruction.

If there is any operation by which that cartilage can be pushed outward and made to grow fast to something out there which would hold it, then it could not be blown in by inspiration, and that is the object of the operation. The structure to which we can make it grow fast is this wing of the thyroid, because that loose plate lies inside of this one wing of the V, and between the wing of the cartilage and the arytenoid there is a mucous pocket. If we take out this mucous pocket there will be two raw surfaces, one on the outer face of the arytenoid, and another on the inner face of the thyroid, and the immovable body will not pass to the movable body, but the movable body will gradually be drawn to the immovable body, which is the wing of the thyroid. And that is what occurs.

A great many years ago Gunther in his surgery spoke of these different operations which he thought might relieve these so-called roarers, or those animals which have this half-sided or one-sided paralysis of the larynx, and that is the operation he advocated, the removal of this sac, but he did not do it often enough to recommend it. He said he did not have enough confidence in the results of the operation to recommend it to his students, but he did put himself in print as expressing the opinion that that might be a valuable operation.

Fifteen or twenty years ago the operation would have been an arytenoidectomy. They thought if they would take that arytenoid plate out they would get rid of the trouble, but the results of arytenoidectomy were that about 30 per cent of those cases died from inspiration pneumonia, due to the drawing of saliva down into the lungs, after passing by the epiglottis and down in the bronchial tubes and then into the lungs, because the epiglottis tips back on the top of the two arytenoids. When one arytenoid was taken away it left a crack and food would go down, and about 30 per cent of the horses would die of gangrenous pneumonia. About 30 per cent were improved, and some few were apparently cured. Then there was another 40 per cent that were neither better nor worse. That showing did not quite justify the operation.

The removal of this mucous sac seems to do better than any operation that has ever been devised.

In any operation for the removal of the sac we should be careful not to cut or break through the vocal cords. If I break away the vocal cord that cartilage drops down, and that lower corner sticks as a permanent wing into the glottis and endeavoring to pull the glottis tends to raise the corner and obliterate the obstruction. As to the cutting away of the vocal cord, this has in it the musculus vocalis, and the ven-

tricle passes right through this broad muscle and splits it into two parts. The part that is toward the median line forms the basis of the vocal cord, and the other part can be seen as a flat band lying up above when the ventricle is removed. So the vocal cord should be left intact to hold up the other arytenoid cartilage, otherwise it will drop downward and inward and will offer a permanent obstruction to inspiration. Any operation that is done ought not to involve seriously, or weaken, this vocal cord for that reason.

The operation we are going to do today is simply this: We get in so that we can make an incision along the edge of the vocal cord from above, near the thyroid cartilage right down to the edge of the vocal cord, until we strike this movable plate, the arytenoid; then we will cut up around this opening which my finger is in. We will cut down the vocal cord first up around as near to the starting point as possible, and then we will join those two lines, so that we have a cut entirely around that opening. Then we want to gather up the loose mucous membrane and dissect it out. You will say, "Why don't you use the burr?" I want to mention some of the advantages and some of the disadvantages that the burr has. In my opinion it does not do quite the work it ought to do, the burr or thimble fails to do what we would like to accomplish by this operation.

It was invented not so much in the interest of the surgeon as in the interest of the fellow who could not do surgery. He could make an opening and find the sac, and he could screw around there with that burr and pull something down and reach up and cut it off, and then it was all done. The burr does not work well for several reasons. Take, for instance, an uncovered burr like that (illustrating). It was an ingenious instrument. It would take hold of the mucous membrane and get at the bottom of that sac and catch it, and you could draw it down and gradually evert the sac. Very often it did not come until it tore. You were drawing the mucous membrane away from the tissue, and you were producing a vacuum, and if it did not tear, the air pressure would drive it right back in again. Suppose this (indicating) was a milk bottle, and you put the nipple down in and sealed it, and then you reached down and got it and tried to pull it out, it would fly back in. The nipple is like this sac. When you pull it up it will go back. And so it is if you wanted to do away with that trouble, all the inventor would have to do would be to bore a hole through it lengthwise, and have a little tip on the end and go down and puncture the bottom of the sac, and the air would rush through the handle and it would come out. But I do not care to make even that improvement on it, because it will not

do even then what we want it to do, because if we had a hole through it and a tip on the end with a hole through it, so that when you went down you pressed the tip through the mucous membrane, the air would come up through the handle when it was screwed up and it would come out here, but the mucous membrane would not come away from the outer face of the vocal cord. The fingers of the burr will not peel the mucous membrane away from the outer or lateral face of the vocal cord, and therefore the cord will not heal fast to and become a part of the wall. It will always stand out like a shelf, unless the mucous membrane is taken away. And you have to dissect that out.

That is one reason why I do not care for the burr, because it will not help me to remove the mucous membrane from the vocal cord. When you get it clipped off with the scissors you will see that there is mucous membrane on the outer face of the vocal cord, and that means it will fill in the part here (indicating).

Now, suppose you started on the vocal cord and ran down the arytenoid and followed around to the starting point, then you have joined the two ends. Then catch the mucous membrane on the end of the vocal cord with a pair of haemostatic forceps, and then take the handle of a knife, and by putting a little tension on that mucous membrane you can gradually tease it away from the outer face of the vocal cord, and then take a deeper hold, and shorter and shorter holds, and finally reach over and get hold of it on the other side, that is on the outer and upper side of the ventricle until you gather the tops of the pouch together in the forceps, and by taking one after another, pretty soon you can get your finger around it and the whole thing is out, and when it is out it is just like the finger of a glove. Sometimes we tear it a little, but it comes together, and you see it is all there. It makes no difference if you have torn it. Then sponge the blood out and put in your tube.

Now, shall we operate through the crico-thyroid ligament in the crico-thyroid space, or shall we go from the thyroid clear down to the first ring of the trachea? I would not myself dare to operate through the crico-thyroid ligament alone for this reason. Those who have operated on many cases have lost some cases with traumatic or inspiration pneumonia. You will see in the American Veterinary Journal a report of twenty-two cases operated on by Dr. Williams, two of which died of traumatic pneumonia. That is one out of eleven, which is nine per cent. No horse that has had this operation done on one side swallows perfectly for a while. For three or four days there is leakage. The side operated on is swollen, and there is a space through which saliva may become mixed with

the food and pass down into the windpipe. There should be some means of escape for that saliva. If you cut a small opening in the larynx there is less chance for it to get out than with a large opening. In my opinion, an opening down from the thyroid down to the first ring of the trachea, cutting through the cricoid, gives you so large an opening that you ought not to have any saliva in any damaging amount, at least, passing down into the lungs.

I have records in the hospital of something over four hundred cases of this operation since July, 1909. A good many outside, also, but I am not counting those. I have complete records of over four hundred cases operated on for hemiplegic laryngitis, for diplegia or double-sided paralysis, and we have not lost any yet from traumatic pneumonia, not one. There have been a few horses that have had a little bronchitis following the chloroform, but I get that bronchitis following chloroform for anything else, following quittor, occasionally. It is not serious. A little increase in the respiration for a few days, a little increase in the blood pressure, and a little increase in the rapidity of the heart; that is all it is. It passes away, and we do not think anything of it at all. But here are over four hundred cases, and not a single case of traumatic pneumonia, although they have been dribbling a little saliva mixed with the food for a time.

What could be the disadvantage of cutting the cricoid? Those who do not do it that way say they do not like to cut the laryngeal cartilage for fear they will start a chronic chondritis or thickening, that leads occasionally to great thickening of the branches of the thyroid and the arytenoid cartilages and the cricoid. All those cartilages, but especially the arytenoid and cricoid become three times their normal thickness and bent to form a curve, so that when you move one out you bring the other in; and those things are not operable. There is nothing to do except to put a tube in the horse's wind-pipe. If it was my experience that cutting the cricoid ever resulted in that, I would quit it, but I have had no evidence in these four hundred cases that it ever has started any such thing as that. I think where we do get chondritis, it is more apt to come from infection within the laryngeal sac, where the whole outer face of the arytenoid and the inner face of the thyroid has been traumatized than from that little dissection of the cartilage at the point where the cricoid is thickest.

So that the longer opening does not produce chondritis, and it is a safety valve and an escape for saliva and food which by the other operation occasionally and too often is followed by traumatic pneumonia.

The diagnosis ought to be simple enough. It is an inspiratory sound. Expiration is perfectly noiseless. If the horse has an obstruction in his nose, such as a foreign body, or as catarrh or anything of that kind and he makes a noise, he makes it both on inspiration and expiration. You may have to drive the horse a little distance before you begin to hear the sound. He will begin to whisper, and then the sound will increase. It is all on inspiration. Put your hand up to his nostril, and when you feel the air against your hand you do not hear the sound, and yet the expired air is as strong from one nostril as from the other. And when you listen to his lungs you do not hear any more vesicular murmur in a horse than in a horse under a little bit of exercise. I do not know that one breed of horses has it any more than another. It was for a long time thought that the English thoroughbred was particularly predisposed, but I think we get as much among common grade horses and mules as among English thoroughbreds. Maybe I ought not to make that statement, but it frequently happens. We do not have enough mules in my state for me to judge about mules, although I have operated on a good many. The man who owns a horse that is worth $150 does not mind as long as the horse can do his work. The hunter, however, has to carry the weight of his rider and has to jump fences and he has to go on soft ground and do it for hours at a time, and it is an awful task on the lungs and heart and if he has any dyspnoea at all it is bound to show there, but draft horses can move along with a heavy load at a slow pace, and such an animal is really not exercised very much, and he may not be exercised until he makes some noise, and even if he does make a little but can do his work, his owner is satisfied.

What are the results of the operation? The best results are in the thin throttled horses. You ought to get about 70 per cent of cures there. I mean seven horses out of ten after proper care for a proper period of time, when put to work make no noise after five months or six months, because it often takes five or six months, and usually more months before the cicatrix and granulation tissue have been contracted down to the final point.

Many owners will write in and say, "Used my horse for the first time; very much improved; a little hoarse. May have a cold. Would you care to see him?" I say, "No, I don't care to see him. I expect the granulation tissue is still spongy and soft, a red scar and not a white pale scar." I say, "That is a little bulky now and it will take five or six months from the time of the operation before the scar has shrunken down and hardened and drawn the arytenoid farther and farther out to the inner face of the thyroid, and it will take five or six months

for that cicatrix to reach its permanent stage, and you write me in five or six months and I can tell you whether it is necessary to do anything." And usually I hear favorably from them when I hear at all, but usually I do not hear at all. When it is all right he does not write, so I usually have to write to him.

About 70 per cent of the horses after five or six months make no noise under the greatest amount of exertion of which they are capable, and remain sound. That is 70 per cent. About 20 per cent are improved. That will bring it up to 90. By "improvement" I mean that the horse after running a mile at a certain gait will not have to slack up for want of wind, or in a hard hunt drop back where he does not belong, and thus shame and embarrass his rider, because a man who has had a good horse that can keep up, would hate to have him drop back among the inferior animals, horses that cost three or four hundred dollars.

Improvement, then, is a horse that still makes a noise, but apparently is not distressed; a horse which has all his speed and endurance, but which makes a noise. That is not a cure, because the noise is the thing that the man does not want to hear. That horse is improved but not cured, because he has still some noise left. About twenty per cent of them are improved. The speed is improved, the endurance is improved, but still there is some noise there. But about one-tenth of them are no better off after the operation or are even worse. We do not expect any deaths. So our expectations are, in the case of a thoroughbred, 70 per cent of cures, 20 per cent of improvements and 10 per cent no better or worse.

Now, among the draft horses, the big Belgians, the grades and the Percherons, especially those with a thick throttle; if you get 50 per cent of cures I believe you are doing well. I know I get just about 50 per cent of cures, and I get about 25 per cent of improvements where the horse is better, makes not so much noise, does not choke up, can do his work, owner satisfied; that brings it up to about 75; and then there is about 25 per cent that will be worse. That is about one out of four or five will be worse. And then those big fellows that are so prone to have the entire larynx thickened up, until it has a big, bony lump at the upper end of the trachea. About 50 per cent of cures and 25 per cent of improvements; so that the operation is worth while, and about 25 per cent that will be no better, or worse, and probably the majority of those will be worse.

We can do the operation under chloroform. What about doing it without chloroform? Just ride the horse in, give him an injection, pull his head up and stand under it, and you can

do it. Even on an ordinary sized horse I can not do very much down this way (indicating). I am looking up in the air and looking up from under, and the horse himself seems to prefer it the other way. Every time you move, that dislodges or disarranges him, so to speak. I cannot blindly put my finger up with a probe, and put something in or cut something off with my eyes shut. I do not attempt to do it. I have stood by and seen fellows do it with what they thought was success. I have some photographs of horses which were killed, where the operator never got into the ventricle, especially where he used his finger. I have some photographs that show the old cicatrix following the use of a thimble. They were supposed to scrape away the mucous membrane where the laryngeal saccule is open, and there were a lot of scars all around. A fellow with a metal thimble on his finger has no feeling, and cannot tell whether he is in the saccule even if the thimble had the power to remove the wall of the saccule, which it has not.

What about firing? I never was brave enough to attempt it. I am too much of a coward to put a thermo-cautery into the larynx of a horse, simply because I know the effects of burning the wall, and I know you have got to be very careful with the larynx of a horse, and to have the least bit of an amount of heat in the larynx. The results of stable fires and in the line of hot air and smoke show what heat will do in the interior of a horse's larynx. Then I know that a horse's tissues differ so much with respect to the same amount of heat. Suppose you line fire one horse and then line fire another horse, do just the same depth of line firing, as near as you can. In the one case there will be no swelling and you will think that you did not fire deep enough. In the other case the effects may be much more pronounced and you will get a little sloughing. The reaction in the one case is great and in the other case is small.

And the same way with subcutaneous injection. You put an injection in, put a sub-tendinous injection in at the point of the shoulder, and you will get quite a big swelling in one case, and in the other case just a little lump at the point of the injection. In horses' tissues there is a great difference in the amount of the reaction.

And so it would be with the cautery. You put the thermo-cautery down into the sac and hold it there for a certain length of time. You may only get a necrosis of the sac where it came in contact with the cautery, but you may also get an extensive necrosis, and great thickening of the cartilage elsewhere, permanent in character. Or suppose you fire through the cartilage, and the arytenoid will drop down, or you may

get a very rapid and intense oedema of the lower portion of the larynx and may have to put in a tracheotomy tube down lower. McKillip has taken it up, but he does not usually publish things that are not favorable, so you may not hear anything about it, but it will have a short, quick death. I prophesy that in a little while you will not hear anything about destroying the laryngeal saccule.

I would like to know if anybody here ever saw a right-sided roarer in which the left side was perfectly sound? I never have. Of course, we can cut the right recurrent laryngeal nerve and produce a right-sided roarer, but it is very seldom involved by traumatism. I chloroform the horse and turn him on his back and open up the larynx and look down in, and he is completely under the chloroform, and the entire arytenoid cartilage drops. I cannot tell for sure which side is paralyzed, but I kneel down and start on the left side. I have examined him before and I know he has an inspiratory dyspnoea, and I have diagnosed the condition as a paralysis of the abductor muscles of the arytenoid. I go ahead and operate on the left side, and by the time I have got through with the left side he has usually come out of the chloroform enough so that he begins to use his arytenoid. The left side is perfectly quiet or faintly moving, while the right side is moving widely in and out. If I did not chloroform him the arytenoid would have been moving under my finger on the left, and when the arytenoid went out it would exercise a little abductor pull on the finger and pull my finger over on the other side.

To estimate the strength of the abductor pull on the right side I could tell readily that the right cartilage pulled my finger more than the left did, and therefore, the left cartilage was weakened. But I do not wait for that because experience has shown me that it is always the left side. Almost all cases are left-sided, and in about one case out of twenty-five or thirty the right side, while not absolutely paralyzed, does not seem to me to move as vigorously as it should, so then I operate on both sides, so that I only operate on both sides in about four per cent of the cases, in one case out of twenty-five, about four cases out of a hundred, about sixteen or seventeen out of four hundred where I have operated on both sides, where it seemed to me that the right side did not have the amplitude of movement that it should have after having operated on the left side, and to make sure I operate on that side.

Then we put the tube in. I like the ordinary tube. It is introduced between the cricoid cartilage and the first ring of the trachea. It has a branch which runs down, with a piece of rubber tubing on the end to permit the horse to bend his neck. This has a long, soft, rubber tube and it is stiff enough

so that it straightens the tube, when it comes up against the larynx, without damaging the tube. This is for draft horses. A draft horse is meaty, and between the surface of the skin and the center of the lumen the distance is greater than in the thoroughbred. So I have two sizes of tubes, and by size I do not mean diameter, because three-quarters of an inch is sufficient for a horse, but only a certain difference in the thickness of the neck, the distance between the skin and the center of his trachea. In a thick-throttled horse that wants to be a little longer, and in a thinner-throttled horse it wants to curve a little.

That is introduced into the lower commissure (the speaker illustrated). This will just go in smoothly and straighten out again. Then I cut that edge off (indicating), which keeps it soft. It is stiff enough to keep it in place. This (indicating) is an incision where it does not fit closely around the neck, but the saliva which escapes through the glottis and passes down through the larynx will go into the elbow around down through the neck and does not run into the lungs.

How will you fasten it in? Carry the piece of tape through the skin, across the incision and through the skin on the other side. Take the center of the tape, push it through that little hole (indicating), pull the ends tight and tie the ends together. When we want to take it out, untie the bowknot, take the tube out without taking off the tape (illustrating). The tape only fastens into the sub-cutis. It does not pass through the sterno-hyoid and thyroid muscles, and lie against the rings of the cartilage, the perichondrium, but it only involves the sub-cutis. You have those muscles between the suture and the trachea. Such a tube is not always needed, but for safety's sake we put it in immediately after the operation. We keep it in three or four days, and we do not know when we can take it out, but on about the third day it may be taken out in the morning and left out that day. We have a man listen to him every once in a while. When first taken out the edges of the glottis will be apart, but soon they will soften up and come together, and during the night he may draw a little air through it. Then we slip the tube in that night and take it out the next morning. If he does not need it the second day we do not put it in again. After three or four days we take it out in the morning, and if he does not need it we put it in again for the night. Take it out the second morning, and if he does not need it the second day, leave it out.

When you operate on both sides in a draft horse it will be better to leave it in for about ten or eleven days, eleven days at the longest. This was in a horse where both sides had been operated on. In from three and a half to four weeks the

wound will be entirely closed. My directions are that the horse shall not be put to any work for six weeks after the operation. The seventh or eighth week he ought to have light work, that is the lightest kind of draft work, or work at a slow trot, a saddle horse should work at a walk or trot. At the end of six weeks they can usually resume their usual work. Do not try to form a final judgment until the operation is from five to six months old, and then you will know what you have done.

After the horse has been operated on two or three months, if he comes in with a bunch of other horses, and if you don't remember whether you have operated on him, there is only one way you can tell whether he has been operated on. I have not a good memory for horses. They come to me as horses, and I just see a horse once, and then I see him on the operating table, and I operate on him and make a record of him, and I may see him a few months afterward or a year afterward, and somebody will say, "You operated on him. Who does he belong to?" "Well, I did operate on him." That is all I remember unless he is a noted horse. I have had to go over and find out whether that horse has been operated on. I remember once we had fifty-two hunters in a ring, and one man wanted to throw out a hunter that had passed beautifully over the jumps, simply because he said I had operated on him about a year before that. I had operated on several horses for this same owner. So I had to go up and tell whether he had been operated. You cannot find the scar unless you shave it, but you can take the skin, and if it is bound down right along the line of the scar you can pull it up loosely on the side, but right there it is bound down as if it was sewed along that cicatrix. And that is the only way you can tell. We have to shave the neck in order to find the scar. So it does not leave any permanent blemish of any sort.

Preparation for the operation is simply this: The horse ought to be put on short feed for a few days before going into the hospital. He comes in and I give him a bran mash, I let him stand up during the night if he wishes, and in the morning he comes in without any food or water, and I always do that before I chloroform him. I just put him on short feed, and on the day of the operation he has nothing to eat or drink during that morning.

After the operation how soon to feed? Whenever you have a horse under chloroform or chloral, especially under chloroform, there is frequently a temporary paralysis of the esophagus which may last two or three hours. During this time if a hungry horse is allowed to pick up straw he may choke by straw lodging in his esophagus, so we put a muzzle on this horse and let him get up voluntarily when he comes to. He

gets up when he pleases, he swings about on his sternum first, he looks around, and in some cases he will get up and fall. We would not give him anything for four or five hours after the operation. His box should be level. The box should be shallow and should be placed down low. Let him eat his hay and drink his water from the level of the floor.

DISEASES OF THE FOOT

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According to the printed program I was to have discussed "Diseases of the Foot." Of course, there are many of them, and it is only my desire to talk about those that you wish to hear me talk about. I would as soon adopt any suggestions you make. What shall I talk about first? Quittor? We do not have very many quittors. For the city practitioner "quittor" and "street nail" and penetrating wounds in the bottom of the foot would be the most important things we can talk about.

A member: We have some quittors through this part of the country from old wire cuts.

Dr. Adams: I will speak about quittor or necrosis of the lateral cartilage, which is the more expressive term. If you read in the old veterinary books you will find it spelled "q-u-i-t-t-e-r." Now it is q-u-i-t-t-o-r. They used to describe it as a chronic suppurative process of the skin or of a tendon or of the lateral cartilage, and therefore, they divided it into cutaneous quittor and tendinous quittor and quittor of the lateral cartilage. Nowadays we do not speak of the cutaneous quittor. We speak of necrosis of the skin or suppuration of the skin; we speak of suppuration or necrosis of the tendon, and quittor is reserved for a chronic suppurative process of the lateral cartilage. Just a word or two about the lateral cartilage and its relations. I suppose that has been taken up in the previous hour, so that I will run over it only hastily, not as an anatomist but as a surgeon, and speak of its relations.

The lateral cartilage is an irregular four-sided plate of cartilage, attached below and in front to the wing of the os pedis, and rather thick in front and thinning out behind and above. If you take the three phalanges you will see from the third or terminal phalanx that we have a cartilage here attached below to the wing of the os pedis, and extending upward and backward, rather thin along the upper margin and rather thick below, where it attaches to the wing of the os pedis.

I want to call your attention to the fact that the cartilage has a firm attachment, not only to the os pedis, but to the lower end of the short pastern. It is directly attached to the lower end of the short pastern so that a little movement of the short pastern is transmitted to the cartilage. It has tendinous fibers which are inserted into the periosteum at the lower end and anterior face of the short pastern. At this point (indicating) we have the lateral ligament of the pedal articula-

tion, passing into the substance of the cartilage, and just posterior to the lateral ligament there is the suspensory ligament of the navicular bone, which is shown at this point (indicating), the capsular arrangement attaching to the whole posterior border of the navicular bone and passing upward across the entire length of the short pastern and inserted here in the periosteum on the anterior face and lower end of the long pastern. That is the suspensory ligament of the cartilage.

Its fibers also pass into the cartilage at that point (indicating), and when the foot is dorsally flexed, the third phalanx, a good deal of the tension is put on the suspensory ligament of the navicular bone, and a little also on the top of the lateral cartilage.

This cartilage is covered on the outer face by a plexus of blood vessels known as the bulbar plexus of veins, here shown quite well.

Quittor, then, is a necrosis of the lateral cartilage. In the city the initial inflammatory process is usually located in the velvety tissue of the sole, and therefore any sole that is so shaped, or any horse that is so shod that the wing of the sole gets too much pressure, bruises the velvety tissue that passes down into that portion of the sole, and we have an exudation of serum and a softening of the horn, sometimes blood clots, and even though there be no break through the horn, even though the horn has not been pared down to the quick, and there is no crack reaching the lower ends of the fleshy leaves, yet suppuration frequently occurs. How is it that such a suppurative process can occur deeply in the tissue, without the infecting germs passing to this tissue directly through a broken integument? You know that germs are being taken in through a multitude of openings, through the nares and the pharynx and the digestive tract, and they are introduced through minute abrasions of the skin, so that every normal being has in his circulation all the time all of these common pus-producing germs. Ordinarily the defenses of the body, the lymphocytes (leukocytes) or the white blood corpuscles and the destructive action of the sera of the tissues soon kill them off, or weaken them so that they are destroyed in the bodily economy; but if some of these seeds happen to fall on fertile soil, they are often sufficiently vigorous to grow in spite of the defenses of the body and to produce pus.

So that an animal with a bruised sole, where the sole is unbroken, may develop a suppurative process by some of these germs that are normally circulating in the system reaching the exudated lymph and extravasated blood, and then beginning to grow and form pus. When pus forms down in a branch of the sole it can readily pass up between the horny and the

fleshy leaves and attack the lower portion of the lateral cartilage. The cartilage itself seems to have a rather poor blood supply, and in the presence of ordinary pus-producing germs is apt to break down and form local abscesses without much pus formation. We might call it an ulcer of the cartilage. The dying cartilage turns pea green in color, and does not as a rule become entirely separated as an island by the suppurative process, but maintains its connection with the cartilage above and anteriorally, so that when you cut down upon a piece of pea-green cartilage, you do not find it loose, but attached on one side.

City quittors originate from suppurating corns and the quittor or necrotic process is low down in the cartilage near the attachment to the wing of the os pedis. On the contrary our country quittors originate through abrasions in the skin at the coronet or above, from the horse tramping upon himself, from a wire cut which goes through the skin down to the perichondrium, and then infection directly attacks the cartilage in its upper half. That is the usual country quittor.

If the suppurative process in the cartilage is left to itself what occurs? It seems that the suppurative process in the lateral cartilage travels naturally from the posterior part of the cartilage forward. It seems as if the posterior portion of the cartilage has greater vitality, a greater power to resist the effects of pus-producing germs, than the anterior has, so that it travels naturally from behind forward.

The symptoms, of course, you know. You have a swollen coronary band, sometimes a little and sometimes much lameness. The lameness is not very pronounced, so long as the suppurative process remains confined to the cartilage, but frequently the process invades the deeper structures. Any suppurative process of the cartilage in the anterior one-third is always apt at nearly any time to pass into the pedal articulation. It usually does it just behind the lateral ligaments of the joint, and in front of the suspensory ligament of the navicular bone.

Therefore, if you have a quittor in this region, and you are inclined to curette it out or scrape it out or slough it out with any escharotic or with a poker, you must remember that you are very close to the synovial sac of the pedal articulation in this particular region. It is not a safe region in which to use an escharotic. It is safer to use the firing iron, because that produces a dry eschar and tends to seal up things, and you can always regulate to a certain extent the destructive action of your firing iron by noticing how much heat you have and by limiting the period of contact; but when you use an escharotic

like zinc chloride, or bichloride of mercury, its action is to disintegrate, and it works into the tissues, and you never know exactly how deep any given amount is going to work.

So that in this particular region it is unsafe to use an escharotic because you are so close to the pedal articulation. Many horses each winter come into our hospital in Philadelphia that are lame with quittors in the anterior portion of the cartilage, in which some escharotic has been used; and when we take away the horn from this region and lift the coronet, we find an opening directly into the pedal joint, with synovial fluid coming out, not clear like olive oil, but cloudy and sometimes greenish, indicating an infection of the pedal articulation, which is a very serious condition.

There is one danger point; low down, three-quarters of an inch below the perioplic band, just posterior to the lateral ligament of the pedal articulation and just anterior to the suspensory ligament of the navicular bone. And if we are taking out the entire cartilage we must be careful not to remove all the cartilage at that point, but leave a little thin film, as thick as a piece of cardboard, over the joint at that point. A little farther back, sometimes the escharotic gets into the bursa, the navicular bursa, and produces serious lameness.

Now we will speak about the operative treatment. There are various advocated treatments. I think, as long as the horse is able to work and is only a litle lame, we are not justified in doing a radical operation which will throw him out of service for a minimum period of four weeks. I think we are justified in attempting to scrape out and clean out the region and keep the horse working.

My method of doing that would be somewhat like this: Suppose we have found upon the coronary band a fistulous tract, that there is a little pus coming out at times, but the horse is not very lame. Take a probe like a lead probe or metal probe that can be bent, a round probe, and pass it carefully down and find out the direction of the tract and how deep it is, catch the probe with the fingers at the surface and draw it out, and then make a nick in the wall at the lowest point. The object is to go through the wall at the lowest point, curette it ou, scrape the granulations out and cut out his pea-green cartilage. One gets a perfectly clean tract through which he can pass a little threaded tube, and then with a bandage on the outside, the animal can continue work. It frequently happens that granulations fill in quickly, and the animal goes on, losing no time whatever.

A little modification of this is to proceed as before up to the insertion of the probe, and then with an ordinary gouge make a round hole which will admit the end of your little

finger, down through the horny leaves into the fleshy leaves, to the end of the probe, so that the end of the probe can be brought through. Then take a little curette and introduce it from above and scrape the region on all sides. As you scrape inside be a little more careful especially in this region, where you might get through the cartilage and into the coffin joint. When everything is crisp and the cartilage that comes under the touch seems like normal cartilage, I would simply clean it out with a little peroxide of hydrogen. Iodine, of course, being one of our best antiseptics, one might clean it out with a little tincture of iodine or a little Lugol's solution of iodine. Then I would take a little cheesecloth, a little wick of it and pass it down through and draw it out and cut it off half an inch from the surface. That wick can be soaked in tincture of iodine or Lugol's solution of iodine or what I usually use is iodoform dissolved in ether. I do not know which is preferable, but I usually take one part of iodoform and ten parts of ether by weight, wet the gauze with it and draw it through. The ether soon evaporates and the iodoform is left in very finely powdered condition in this little wick, which acts as a nice drainage tube. Then to keep the poultice from undergoing the effects of capillary attraction I cover it with sterile cotton and put a bandage around the foot, with the shoe on. You know how to keep a bandage on an uneven surface like that. The tendency would be for the bandage to slip off. Take some strong material, about an inch and a quarter wide, like heavy muslin, and pass it around the center of the foot, over your cotton and make a half-tie at the heel just above the branches of the shoe, which ought to be a little long. Then carry the second wrapping around the top of the dressing, and then go progressively from the top to the bottom. Then go up to the top and down to the bottom again.

How do you keep it from slipping? If the quittor is on the outside of the foot, we have a hook, the shank of which is driven in between the shoe and the foot, at the inner toe. It is a broad hook, and every turn of the bandage goes under the hook, which keeps it from slipping up over the coronary band. When all of the turns have passed the heel, the end that is free is run once or twice around the bandage, and they are tied together and the bandages are smeared with an adhesive to keep them from slipping. They lap over like the shingles on a roof; so that the horse which has a tendency to stand on his toe or on his heel does not tear the bandage off. That dressing has to be taken off every three or four days and the wick replaced very frequently, and that is all that is necessary after curetting.

I do not believe it is safe or good procedure to use escharo-

tics to destroy the cartilage in preference to the curette. I think the curette is better. Next to the curette comes the little poker heated to a good heat, but of the two I prefer the curette.

That is one of the treatments that sometimes or frequently results in keeping these animals working and in restoring the the foot to its normal or nearly normal condition, without radical operation. But most of the quittors that come to us, for instance in Philadelphia from the city, have passed that point. As long as the animal was able to work he was worked, and when he gets so that he cannot work he is brought in. Frequently the coronary band from the inside or outside toe, clear back almost to the heel, is sloughed, fistulas have formed, the perioplic horn and the middle layer are separated to a certain extent from the coronary band, which is raised up out of the groove. We then have to decide on some form of extirpation of the cartilage. When you go so far as to take away a section of the wall and get under the coronary band, you might as well take out all the cartilage. You cannot tell by looking at or feeling of the cartilage whether it is infected, and a partial resection of the cartilage is not as good as a complete resection. Just as in penetrating street nail, when you have necrosis of the tendon beneath the navicular bone partial resections are not advisable. A tendon that looks crisp and hard and yellow in color and looks normal may be infected and in a few days it may soften. So that partial resection of the cartilage and of the perforans tendon beneath the navicular bone are not really advisable.

We have a great many quittor operations. Some of them are complicated, or comprehend certain divisions of the coronary band, so that the cartilage may be more easily exposed.

I want to speak first of one of these operations, of which a great deal has been written in the last few years, and which in my opinion and in that of many others is the worst operation that was ever devised, and that is known as the Bayer operation for quittor. Professor Bayer is alive yet. For many years he was professor of surgery in the veterinary school at Vienna, in the Austrian Veterinary School, a very lovable old gentleman, and a great credit to the profession. He devised many years ago the operation for quittor which has been given his name. It consisted in making a U-shaped incision involving the skin above, the fleshy leaves and the coronary band. It makes a nice looking operation, but the results are poor. The blood supply has to reach the flap entirely through the narrow isthmus or neck at the top. The pressure of the bandage compresses the vessels, and the result is that this flap always dies. Having taken this cartilage out beneath

there is a space there, and blood and serum pass into it. Again, the pressure of the dressing forces the coronet in, and if healing is fairly good we have a deformed quarter. But more often those secretions become infected and an abscess forms. It is a very unsatisfactory operation to me. I tried faithfully to carry out Professor Bayer's directions, and I felt it must be some fault of technique on my part; so that four years ago, or in 1911 when I visited the Vienna School again, I found Professor Schmidt, Professor Bayer's successor, and I said, "I am glad I am here. I am at the home of the Bayer operation, and I want to see it done." He smiled and he said, "We do not do it." I said, "How is that?" "Why," he said, "we have never done that operation since Professor Bayer resigned." I said, "Why?" He said, "It was very laughable. It was a pet operation of Professor Bayer's, but it never gave him any results. It was always a joke around the place whenever Professor Bayer did that operation. He did not get results. He only persuaded himself that he got results. It was a jest and a laugh among the students." I said, "That makes me feel better." "No," he said, "it is an operation that cannot be done properly. So forget about it. That is all. Just forget about it." And that was Professor Bayer's operation.

Whenever the suppurative process is in the middle, whenever there is a quittor and the heel is not distinctly swollen, when the posterior third of the cartilage is about normal apparently, I would advise the operation of Moeller, Professor Moeller, who was professor of surgery in the school in Berlin many years—I think he is in private practice in Berlin now; but he was a good horseman, which means a good deal when you are talking about Germans, because you seldom find a German who is a horseman in the sense that an American uses that expression. They go on the wrong side of the horse and lead it with the wrong hand, and they are always in front of the horse, and they will come up to a horse and punch him in the ribs and say, "Get over." I wonder there are not more of them killed as a result of their methods of handling horses.

Well, Professor Moeller was a good horseman. You would think he was an American if he did not have that name Moeller, which is a German name. And he was a practical man, because he always had a large practice. He was not one of those professors that has a sort of an idealistic picture of the animal and writes all about it from his study, but he was a man who was right up against the horse, and he taught his students things, and the way to do things that he had learned by experience were the best ways.

We will suppose the suppurative process is in the middle, and the anterior third of the cartilage. There I would advise

the operation of Moeller, and Professor Frick, his assistant, who is now professor of surgery in that well-known school.

It consists in removing half of the circular piece of horn from the wall, beginning at the anterior portion of the cartilage and running down toward the plantar border, and up in front of the buttress, making a groove, and then using a little iron pry like a chisel, bent a little at the end. This groove is made down to the fleshy leaves, the pry is introduced, and the piece caught with a pair of blacksmith pincers and torn off. You can control hemorrhage by putting a rubber tubing around the pastern in this region and just above the fetlock joint. The pastern is perhaps a little better. Then take a knife and cut through the pododerm along the lower border of the coronary band. Next take a sage knife or convex scalpel and cut up underneath the skin and keep the finger on the skin so that you will not cut through it until you get up to the top of the cartilage. Then pull the coronary band out with a pair of retractors, pick up that plexus of blood vessels that lies exposed, and dissect them off the surface of the cartilage. Afterward you can shave from below upward, shave the cartilage until it is practically gone, not until you feel the plantar cushion like soft tissue, but until it is just covered by a thin film of cartilage. In other words, you go clear down to the perichondrium, which covers the inner face of the cartilage. If you do not dissect off that little venous plexus inside of the flap your knife will not cut well, but if you will pick it up, cut the plexus parallel with the incision below and dissect it up until you see the cartilage, then it shaves, and with a sharp sage knife there is no trouble in getting it away. But in the region of the lateral ligament low down forward never try to get all the cartilage. Leave a film at that point as a protection to the joint.

With this operation we cannot get around the heel, but we choose this operation when the heel does not seem to be much involved, when the suppurative processes are in the middle or anterior half. That is the operation of Moeller and Frick.

You have cleaned the cartilage out, and what shall we do next? We ought to put some non-absorbable material in to take the place of the cartilage and to hold the band up where it belongs, until it is stiffened by new horn, and also to take care of the secretions, and for that purpose we use ordinary sterile cheesecloth, which is cheap. Take a little wad as big as a handkerchief and put it together. I usually take a 10 per cent solution of iodoform in ether, or Lugol's solution of iodine, take the cork out of the bottle and tip the bottle up until by capillary attraction it wets the whole mass. Then I

push that up under the coronary band until I have the space from which the cartilage has been taken just lightly packed. Be careful to let the lower end of the gauze hang down and cover the surface of the fleshy leaves. Squeeze the cotton out of a solution of bichloride, 1 to 1,000 is enough, and make a pad first, that will cover this area and not run over the coronet at all. What we want to do is to have a pillow of gauze below the coronary band so that the subsequent dressings will not press too strongly on the coronet. A good stiff pad of cotton below, so that when the second pad goes on, the coronet does not get too much pressure; run the next pad clear up to the fetlock and away down over the foot, just wrung out of bichloride solution, and then cover it over with an ordinary bandage, and that in turn with clean burlap and let the horse out.

The first dressing ought to be about four days after the operation, and you take all the dressing out. It will be found soaked through with blood. The blood on the surface is dry, and infection does not readily pass through, because the surface is dry, and it goes into the bichloride solution, anyway, from the surface. About four days after the operation you redress in exactly the same way and you continue to do so about every four days, depending on the amount of damage. If there is very free discharge perhaps every third day. If dry at the end of the fourth day let it go six days, because the less often you have to dress it the better. You continue to pass the gauze up into the region from which the cartilage was taken, as long as there is any space there to pass it into, and always let the gauze hang down between the lower edge of the coronary band and the upper end of the fleshy leaves.

If you are careless about that, if you have failed to pack the gauze up under the band while there is still space there, it frequently happens that the lower edge of the coronary band it will grow fast to the edge of the fleshy leaves, and will harbor the wound secretions, and the coronet will soon swell and get soft, and you have to go in there or it will burst and there will be a little abscess in the region where the cartilage was. That is simply because you have not provided for continuous drainage. You have not continued to pack that little place up and let it hang down, between the lower edge of the coronary band and the wall.

At the end of a week the fleshy leaves have covered the place. It is surprising how soon we will get a light coating of horn over those fleshy leaves. After six or eight dressings, four days apart, the space is filled. By that time we find a quarter of an inch of soft horn on the coronary band, which holds it up in a normal position. The fleshy leaves have be-

come covered, also the innermost layer of the wall, and the horse is usually ready to go to work about the fourth week. Shoe him with a bar-shoe, so that in the region of the affected quarter the shoe does not touch the foot, put a tar dressing on the foot and put the animal to work in about four weeks.

That is the operation of Moeller and Frick, which is, I think, the best operation where the suppurative process is in the middle or anterior part, and the heel is not much swollen.

Sometimes the coronary band at the heel has separated from the coronary growth and the quarter is raised up. We then realize that we have to do our operating in the posterior half of the cartilage to remove it. In that case the French operation of LaFosse seems to me the nicer operation of the two. The quittor that is most often treated by the LaFosse operation is the quittor of flat foot, the one that has suffered with suppuration in the angle of the sole, where the process has gone up onto the fleshy leaves and infected the posterior half of the cartilage.

In that case we can begin at the anterior portion of the cartilage and draw a line downward and backward so that it strikes the plantar border in front of the buttress. Then we thin out with a hoof knife, the angle of the sole and the inner bar. Then we introduce the pry and pry that quarter upward and backward, tearing it from the coronet progressively from forward to backward, and it takes out the whole quarter. The portion of the bar in front is thinned out and the whole branch of the sole is thinned.

The wall is taken off back of my finger (indicating) and we have the coronary band and the fleshy leaves of the bars back to this point. Then we can get underneath the coronet and go right along the lower border of the coronary band, and under the pododerm and for an inch alongside of the frog at the upper end of these fleshy leaves, because this is the bar portion of the coronary band. So that we follow that around above the ends of the fleshy leaves, and for an inch in distance. The knife comes up clear around the heel. In that way we can get at the suppurative process well back in the cartilage.

The operation from that point on is the same as before. Dissect away the venous plexus and shave the cartilage from above until we have removed practically all the cartilage, and be very careful about one thing. The suppurative process has usually resulted from suppuration in the angle of the sole, and the lowest part of the cartilage is involved, so that after raising the coronary band and shaving the cartilage away, we

should push the coronet in and cut the cartilage away clear down to its attachment to the wing of the os pedis. Often the process is down there although the pus is coming out above the coronary band.

Then dress as before, and shoe with a bar-shoe, without pressure on the affected side, pressure on the frog and all around the circumference clear back to the groove on the other side, the affected heel resting in the air practically, but covered over by the shoe below. That is the operation of LaFosse, described many years ago. Its principal value is when the suppurative process is back in the posterior third of the cartilage, and it is necessary to get around the heel.

Those are two quittor operations that when an operation is necessary will serve all the purposes, so far as the curing of the quittor is concerned.

Next let us talk about street nail. The so-called street nail is just a pathological condition due to the penetration of some foreign body in the bottom of the foot. Any penetrating wound in the bottom of the foot that involves the pododerm, the fleshy sole and the plantar portion of the coronary band, or any other structure in the bottom of the foot. All of those conditions are grouped under the one condition of "tread." The object that penetrates is frequently a nail, and the point of penetration is usually the deepest part of the concavity of the sole, where the horn is moist. It seldom, of course, goes through the sole unless it is sticking out of a plank or some fixed object. Then it may pass into the sole.

At the point of the frog such penetration usually strikes merely the os pedis where the perforans tendon is attached to it. A little farther back, an inch and a half or so back from the point of the frog if it goes in vertically, it passes through the tendon, passes through the interosseous ligament, between the anterior border of the navicular bone and the os pedis, and passes directly into the joint. A little farther back, if it goes deep enough, it hits the navicular bone.

We will suppose a horse has a wound in such a region and direction that it may involve the perforans tendon. That horse, of course, is quite lame. He is knuckling over because he wants to relax that perforans tendon. He is tender to pincer pressure in this region, and if the plantar cushion is swollen, this hole in the heel has gone, and it is flat or convex, showing that he has a violent inflammation in the plantar cushion. Then we presume, if we have synovial fluid coming out, that it comes there from the joint, or from the navicular bursa. How may we roughly determine whether or not there is an inflammation, a synovitis of the pedal articulation or whether we have merely a synovitis of this navicular bursa?

Whenever there is a synovitis of the pedal articulation, in other words, an infection of the joint, we have a little light ring-like swelling all around the foot, about an inch above the coronary band, or just a little above it, and a good deal of pain when the toe is manipulated, whether drawn back or forward, and a little ring-like swelling around the foot, indicating that there is infection in this joint, but with infection of the navicular bursa and no infection of the coffin joint, you do not find that ring-like swelling. That is important to remember. A horse comes in lame, with violent pulsation in his digittal arteries and plantar arteries, and he has a light ring-like swelling, then the prognosis is bad, because I know there is infection of the pedal articulation.

We have a nail wound, and suppose the horse is very lame, the wound is far forward, so that we think it has gone into the os pedis or coffin bone. We are then justified in using some remedial measures. Suppose the nail is near the point of the frog, the first thing is to soften it up. This horse's foot is put in a tub, if the foot is dry; if he has had hoof ointment on it it is well to take warm water and get the grease off the surface. The water does not penetrate through the layer of grease. Use washing soap, the kind of washing soap which a woman would use for dirty clothes, and give that foot a good scrubbing; and put it standing in a mud hole or tub, or with wet swabs on his feet. It will take from sixteen to twenty hours to soften up a good thick foot like this specimen, which is hard. That softens up the horn and makes the foot spread and accommodates the hoof which is dry and contracted, to the swollen part within, and gives the horse a little comfort.

Then we thin this entire sole until it springs under the finger, more than you would have it thinned in a blacksmith shop. In thinning a sole for shoeing in a blacksmith shop we do not as a rule take all the dead horn from the surface. We stop thinning before all these little cracks appear; but in this surgical thinning we thin until the horn springs a little under the finger; we aim to thin the horn off here about an inch and a quarter or an inch and a half entirely around the nail hole, used as a center. Thin the horn off here an inch and a quarter to an inch and a half on all sides of the nail hole, and thin it down to funnel-shape. Where the nail has penetrated, thin the horn here to the size of the surface of your finger nail. Then you can take a little curette. The ordinary curette which we have in our cases is not quite the sort, but in some of our cases we have a little curette which is small and a little curved, and we can cut that clear to the bottom. And then I would put in an antiseptic dressing and cover it over. It frequently

happens that you have removed the infection and the inflammatory process passes away.

In cities, especially farther south, it may be well to give the horse a preventive dose of tetanus antitoxin. There are certain sections of country like Philadelphia where such wounds are not often followed by tetanus, but there are certain other centers, particularly in the south, where the tetanus germ seems to be virulent and widespread, and it is not safe to leave such a covered wound like a nail wound, with ordinary treatment. It is better to protect it by immunizing with tetanus antitoxin.

That is an operation which has often been of service where the nail wound is not farther back than half an inch posterior to the point of the horny frog, but when we have a nail wound in the lateral cleft between the bar and the frog, we know it must have passed through the pododerm. If it hit the pododerm it inflames it, damning the blood back in the plantar artery, and it has gone perhaps through a portion of the plantar cushion and strikes into the perforans tendon. This tendon is of mixed yellow elastic and white fibrous tissue. The suppurative process does not tend to limit itself in this yellow elastic tissue. When we have an infection in yellow elastic tissue like tendon tissue or the fascia of the belly, the suppurative process tends to spread farther and farther, and not to be separated by lines of demarcation. It spreads like a rotten spot in an apple, which becomes bruised and infected. The only way to get rid of it is to cut into the sound apple at a little distance from the spot. The same applies to infection of the perforans and other tendons. We have often then to cut away the entire attachment of the perforans tendon to the semilunar crest of the os pedis.

And how far back can we go in making the cross cut? We can go to the middle of the inferior surface of the navicular bone, or we can go as far as the posterior border of this navicular bone. In other words, if our nail has gone into the perforans tendon and infected it, we can cut the tendon off half way between the anterior and posterior border of the navicular bone, or in extreme cases we can go to the posterior border.

We will presume we have decided to resect the perforans tendon. We have softened and thinned the horn, and gotten the horse ready for operation. It may be done under chloroform, or it can be done very well under local anaesthesia, injecting over the digital or plantar nerve. I would advise you in this case, if you are going to use a local anaesthetic, not to take the whole nerve, that is the posterior of the three digital, but go up above the fetlock and take the inner and external plantar nerve before they have divided into the digital. The

reason for that is that the posterior digital nerve which is the largest of the three digital nerves, does not always supply exactly the same area. It supplies most of the navicular region, but sometimes not entirely. Frequently the middle digital nerve supplies a portion of this region. When a horse has a navicular bursitis, and we do the whole operation, cut the posterior of the three digital nerves, the horse continues to be lame until we go up and cut the plantar, and until we search for the middle digital nerve, which is difficult to find because it is so small. So the point I want to bring out is, if you cocainize the posterior digital you do not always thoroughly anaesthetize the region you are operating on. But you can anaesthetize it with an anaesthetic above the fetlock joint, and then you can put on your haemostat or ligature, and cast the horse and secure the leg. The animal has to be cast, and you can do the operation without a general anaesthetic, although it is very much more satisfactory to do it under a general anaesthetic.

And those of you who have not been chloroforming horses ought to start in and get busy. It is simple and relatively safe, and it is so much more satisfactory to the operator, and I know it inures to the good of the patient, because if the horse is conscious he frequently makes a movement when you don't want him to, when your knife is in a place where you would not be able to withdraw it quickly; very often a bad cut is made because of a horse moving quickly just when you did not expect him to move, and when your instrument is in a dangerous region. So that I operate entirely under a general anaesthetic, under chloroform for horses.

The old operation for resection consisted first in thinning the horn of the entire sole, thinning down the horn to the frog until every point could be easily cut through, until every point springs nicely under the finger, and then cut away the substance of the frog. Make a transverse incision, beginning at one branch of the sole, going across the bar, and the frog near the anterior portion of the middle cleft of the frog, across the other branch, across the other bar to the branch of the sole on the other side. In other words, making a transverse incision across the frog at the anterior portion of the middle cleft. But that anterior portion of the middle cleft is such an indeterminate thing.

I think it is better to go by the extreme branches of the sole and let the incision join the extreme angles of the sole. That carries it across the frog. Make your incision a vertical one or at right angles to the direction of the sole itself, down through the frog, and then you may go on the side and take the body of the frog away. That frog is nothing more than

the continued extension of the plantar cushion which happens to be the shape of the velvety frog. You may cut away the body of the frog, and then the bars, and from the ends of your transverse incision you can make a circular incision down to the point of the frog, and take all the pododerm away. That was the old way of doing it, and it is very good, but it is not as good as another way.

This method consists in making a transverse incision as I described, except we do not allow it to go through the frog at all. It goes from a branch of the sole, across the bar down to the frog. On the other side, from the frog across the bar to the other branch of the sole. And then we insert the knife beneath the body of the frog, along one side to the point, and along the other side to the transverse cut, and dissect the plantar cushion or the soft frog away from the under-surface of the perforans tendon. Cut it loose from its base, put a tape on it and pull the soft frog back. Let somebody take the end of the tape and turn the soft frog back, and the whole tendon is exposed as much as if the body of the frog were cut away. We afterwards have a normal frog when healing is complete.

If we cut away the velvety tissue of the frog, granulations will fill in and the velvety tissue of the sole grows up over the granulations, and the new frog that is formed has the character of the sole and not the character of the frog. Back at the stump, the velvety tissue of the frog grows forward and produces horn that is like the original frog, but it mingles with the extension of the velvety tissue of the sole, which is more nearly pure horn, as hard as the sole, because it is produced by extension of the velvety tissue of the sole. Such a frog must be constantly thinned, but if after thinning the sole, the bars, and the horn and the frog, you cut underneath the body and turn the soft frog back, then after resecting the perforans tendon you can let that frog go back in place, and you have a normal frog.

As for resecting the tendon, I do not need to go into any details with reference to that. We have this widened V-shaped portion of the perforans tendon, where it spreads out in this V-shape attached to the semilunar crest, of the os pedis, from one wing, and from the os pedis. If the nail has gone into this region, and that is the part that is necrotic, we can cut that tendon off as far as this posterior border of the navicular bone, but not much farther because then we would cut through the ligaments that we meet in the synovial sheath of the pedal articulation, and we would open the pedal articulation from behind. We can feel the anterior border of the tendon by pressure, and of the navicular bone by pressure, or if we are not certain we make a transverse incision and longi-

tudinal also, and gradually cut through until we find where we are at. Finally we cut the tendon through to the end of the navicular bone, and cut it off and cut it away from the semilunar crest, always being careful not to tear this ligament between the navicular bone and the os pedis, because that is thin and some of the diseased tissue is always attached to it. It is always better to leave a little of the diseased tissue attached to that interosseous ligament rather than to run the danger of tearing the ligament by trying to get it all away. Clean it out with peroxide and put in a veil of gauze with iodoform, ether solution or bichloride pads, and dress it every four or five days or a week. If it remains dry, the diseased tissue fills in from the end. Granulations spring up almost immediately.

After cutting the tendon away, even though the cartilage on the under side of the navicular bone has not been touched and seems to be crisp and smooth, we had better scrape that cartilage away. Then the granulations will spring up over the bone, and will fuse with the granulations springing from the tissues, the fibrous tissue, and we will have the tendon attached to the under-side of the navicular bone; I mean the granulation tissue will be attached also to the interosseous ligament in front of the navicular bone, and to the os pedis itself. The new tissue will never be as strong as the tendon tissue, and we like to gain every possible square inch of tissue that we can get. If you leave the cartilage on, the granulations will not spring from the cartilage, and this new granulation tissue will take no attachment to the navicular bone at all. You will not have as good results as if you scraped the cartilage from the under-side of the navicular bone.

The operation itself and the thickened granulation tissue that attaches the end of the tendon to the end of the bone interferes a little with the dorsal flexion of the third phalanx, and the animal is a little stiff in that joint. Gradually by contraction of the tissues the fetlock is forced forward and the animal stands stiff in the pastern, the heel is raised by contraction, and the animal gradually acquires a club or upright foot. It sometimes happens also that under extreme work, putting strain upon this granulation tissue, in the case of the hind foot, that that new formed tissue, cicatrical tissue, is unable to stand the strain. It breaks, and then there is a partial dislocation of the lower end of the short pastern down over the surface of the navicular bone. This is an operation to save life, and frequently renders the animal serviceable for slow work for some years, and therefore it is justifiable; but we must never expect speed in horses which have had the operation done. It would be valuable for a breeding mare or the stallion, but we need not expect speed in these animals.

Side Bone: Side bone is an ossification of the lateral cartilage. It is almost physiological in some species of horses. Take the Belgian and some of the strains of blood in the Percherons, and you will find two-year-old animals with pretty well formed side bones, which have never been off the farm and have never had a shoe on. You have seen them on the hind feet as well as the front feet. When the ossification is bred into an animal there is no use in trying to do anything with it. But suppose the cartilage is not ossified when the animal is four years old, and he is sent to the market, and he goes to work on hard streets. He develops laminitis, an inflammation of the fleshy leaves back of the quarters, where the os pedis is soft, inside of the wall. He gets this chronic condition, associated, perhaps, with corns. The fleshy leaves are involved. There is an ostitis of the wings of the os pedis, and this ostitis passes up into the cartilages, where they are inserted into the wing of the os pedis. There develops a chronic chondritis, which is followed by deposits of lime salts in the cartilage. (Addressing Professor Murphey.) Professor Murphey, you do not regard this as a true ossification, do you, or is it rather a calcification? Do you find the bony Haversin canals running up into the cartilage?

Professor Murphey: I have not made any sections of that. A few of them we have X-Rayed, and the bone seemed to be normal. In some cases it was uneven on the surface.

Dr. Adams: We will not take up that point, as to whether the cartilage at its attachment to the wing of the os pedis changes into true bone, bone consisting of cells with the Haversin canals between, or whether it is rather a calcification of the cartilage. It is not entirely abnormal in that it does appear in colts, that have had no such traumatisms at all, so that it to a certain extent is transmissible, and to certain extent it is hereditary. It occurs rather in the upright or deep feet with relatively vertical or upright wings and outer walls. This is not a disease of the flat foot and the low heel, but of the high heel and the upright foot, and therefore more often in the Percheron does it occur than it does in the Clydesdales.

So long as these cartilaginous plates are elastic and soft and uncalcified or unossified, whenever the weight is placed on the foot, the phalanges sink back and with the sinking back of the phalanges the short pastern pushes on toward the tendinous head, and you have pressure on the plantar cushion, and the cushion tends to become pushed down through the foot. But that pushing down is resisted by the pressure of the frog, through the rubber pad, and this plantar cushion, whose function it is to take the shock away from the joint surfaces when the animal under speed throws its weight down upon the hard

ground; this plantar cushion, squeezed from above, and unable to expand in any other direction, tends to expand laterally. But the lateral plates are in absolute contact with the plantar cushion on the sides, and when the plantar cushion is squeezed above, and possibly from below, it expands laterally, upon the lateral cartilages, which are covered by the fleshy leaves of the quarters. So the widening of the posterior half of the foot depends entirely upon the elasticity of the lateral cartilages, pushed aside, of course, by the expanding plantar cushion, which is a rubbery or elastic body.

When the lateral cartilages, however, are ossified, and when the fetlock sinks under weight and the short pastern rotates backward onto the plantar cushion which it tends to push down, but the shoe or rubber pad prevents, and the plantar cushion expands laterally and compresses the lateral cartilages apart, they being stiff, put pressure upon the pododerm.

Where does the ossification begin? It always begins far forward, where the cartilage is attached to the wing of the os pedis, just about opposite the lateral ligaments of the pedal articulation, and the ossifying process passes upward first and then backward, forming a hook. When formed rapidly it produces soreness in the foot, soreness on pincer pressure, a shrinking on stepping on hard pavements, from the sting that is produced every time the foot is placed on the ground.

Many a side bone begins from an unbalanced foot, where it is not hereditary. You will see a horse walking on the outside of his feet, with the outer branch of the shoe worn thin and the inner calk half an inch higher; the outer calk worn down on one side, and a quarter of an inch higher on the inner corner.

That horse has been unbalanced. When he was newly shod, the outside of the foot struck first, and the foot tipped to the inside. The first shock was received on the outside of the foot, because the shoer did not dress the foot in accordance with the wear of the shoe. If he found the old shoe worn thinner on the outside than on the inner, he should have taken his cue, because he should have known that the outer branch, more worn, was under the part that was too long, and he should have taken away all the extra growth on the outside, until he was able to put a new shoe on which would come down flat, and which would get the same amount of scour on the inside as on the outside.

The result of the outside coming down and getting the sting or hammer on the ground, is to set up an inflammation on the outside of the foot, and it starts a low grade inflammation, which results in ossification of the cartilage. So that the

foot that is too long on the outside and the outer cartilage being the one that is chiefly ossified, is often the starting point for side bone in horses in which side bone is not bred in them.

Suppose he has side bone, what can we do? Shall we give him frog pressure? Shall we put on a bar shoe, or a little rubber pad to give pressure? No. The quarter under the side bone is flattened a little and contracted. We should try to expand the quarter a little. You can do it in two ways.

You can soften the foot, first by soaking, and you can put in the bottom of the shoe a Chadwick spring, which is a steel spring, V-shaped, that lies in the bottom of the foot, and the ends of the spuds or branches are inserted against the bars just in front of the buttress. The point is inserted into the bar in front of the buttress, and then it is laid down on the bottom of the foot, and tar and oakum and a leather sole applied to keep it from dropping out, and then an open shoe. That spring exercises continuous pressure and relieves the tightness of the quarter on the side bone. You cannot spread the heel very far if there is a fully formed side bone, but you can spread it enough to remove this sense of tightness of the quarter against the fleshy leaves covering the lower part of it, so that the Chadwick spring by exerting continuous pressure underneath a leather sole assists materially in making the animal feel comfortable in the foot.

Then the shoe itself may have toes and heels, but it ought to be fitted full, a little beyond the quarter of the side that has the side bone, and the branch a little longer than normal. Preferably we would use a shoe without toes and heels. But if it is necessary for the horse to get a grip on the pavement, they may put on toes and heels, but he ought not to have frog pressure. He ought to have something that holds the buttresses apart without frog pressure, and the shoe should be fitted full along the quarter, and the nails should be carried far forward toward the toe so as not to interfere with the expansion of the quarter, the nails carried around the toe, the last nail two-thirds of the way back.

The branches should be long and fitted full, and in some cases the hoof surface may be beveled slightly upward and outward, so that the tendency is to slide outward. How much bevel shall we give it? That is an important thing. If we give the shoe too much upward tip and the quarter falls on it when the horn is soft, it sometimes happens that the wall may separate from the margin of the sole and we get a separated wall in the quarter. The amount of tip can be nicely estimated in this way. It is a perfectly safe rule. I can illustrate it to you on the foot. If I put a ruler across the buttress, and raise the ruler about half an inch above the horn on that side (in-

dicating) that gives you a tip that you can safely give on the other branch. The amount of upward tipping of the bar surface is estimated by raising the ruler half an inch from the foot on the other side.

Dress the foot so that it will come flat on the ground, have it soft, put in the Chadwick spring or if you have not got it, put the bearing surface or branch of the foot downward and outward inclined (illustrating), an amount equal to raising the straight edge half an inch above the opposite branch, and that animal will travel as well as it is possible for him to travel.

But somebody says suppose we rasp the wall from the coronary band to the bottom. That is a lazy man's way of accomplishing the same result, to rasp it with a hoof rasp from the coronary band down to the bottom, so as to make it thin, and then put on fat or lard or grease and keep it soft; but the trouble is that we sometimes get plantar cracks or coronary cracks, not quite so bad as we would in another foot, because there is so little expansion of the crack that it is not apt to cause trouble.

But it is not necessary to thin the horn, if we keep it soft and have an expansible shoe on, and no frog pressure, which is very important. Keep the foot dressed so that he wears his shoe evenly. Whenever the old shoe that comes off is worn more under the side, that means that we have too much length of horn on that quarter, and it ought to be removed.

Q. You mean, then, to cut down on the worn side?

Dr. Adams: Cut down well on that side and thin it.

Q. Cut down on the side it is worn on?

Dr. Adams: That is the worn side. It is not the weight that wears. It is the scouring that wears, when the foot is first set to the ground.

You must spare the other side. Let the other side grow longer, until the foot comes flat, and then the sides are relatively of the proper length. Whenever the outside of the foot hits first it is relatively too long, and the other side must be allowed to grow longer, so that that foot ought to come down flat.

If a horse without a shoe traveled over perfectly level surfaces, like sandpaper, he would level himself just about right. The part that hits first would scour off, and finally his foot would be brought out perfectly flat, and that would be the ideal way of leveling it. But man puts on iron shoes which prevent wear against the foot, and the iron shoe wears slowly, and where that shoe shows some wear, that is some evidence that that part of the foot is hitting first. With great speed,

the horse always throws his heels down, to get that plantar cushion down to break the shock.

Suppose you take an instantaneous photograph of trotters or runners, a running horse with three feet on the ground, one on the ground just about touching the ground, the toe is always turned up and the heel is turned to the ground, so that the plantar cushion gets the shock. It is like rubber heel shoes worn to break the shock.

But at a slow trot or slow gallop the foot comes down flat and slides along the ground, and it ought to get the same wear on the inside and outside, and at the toe and heel. But if one side strikes first with the shoe on, he will gradually scour that part thin. That is evidence that the wall above that is relatively too long, or that the other side is relatively too short. It often happens that the part that strikes first is so short that we cannot cut it down without getting it too thin. It is relatively too long. The treatment is to allow the other side to grow out until it will hit as soon as or a little sooner than this side. The side that hits first may be too short actually, although it is relatively too long. The opposite side must be allowed to grow until that foot comes down flat.

And in side bone, if the horse has been hitting on the outside, it sets up a chronic low grade inflammation of the outside, which spreads up to the cartilage, and he begins to develop an ossification of the cartilage. He is a pigeon-toed horse. He is a broad-breasted fellow, pigeon-toed, and he swings his foot out and strikes with the outer toe, like all such horses do, and the outer toe side wall is worn thinner than the inner. The shoer noticing that ought to always go after the extra horn on the outside and spare the inside, until when he swings that foot around it comes down flat and wears his shoe uniformly all the way round.

What shall I talk about next?

A member: Take cracks in the wall of the coronary band.

Dr. Adams: Going clear down to the plantar border, complete penetrating cracks. They are more often seen in the inner quarter in a narrow foot, in trotting horses.

The member: We get it lots of time in Iowa from barbed wire cuts.

Dr. Adams: Yes; that goes through the skin and the coronary band, and the coronet has remained open, and thereby the coronet becomes separated, one portion sends down a piece of horn and the other portion sends a piece, and finally that crack in eight or nine months has reached the bottom. What can we do? That is very important, and there is only one way to do it, I think. The trouble is with the divided coro-

nary band. If that coronet would only unite and send down a solid sheet of horn we would be through.

The product of the pododerm is an epidermal structure like the hair of your head, and there is no treatment of the horn which will ever make the crack unite. It is an epidermal structure, and two epidermal structures can lie close together and never unite. If they did, you would go to bed some night and in the morning find the hairs of your head had grown together along the sides. You have to go to the matrix from which the horn comes and get a union of soft tissues, and when that occurs a solid sheet will be thrown down.

So we are up against this problem, to expose a certain portion of the coronary band and freshen it and let it heal first, and then allow the horn to grown down. We begin, say an inch in front of that crack, and make a V-shaped groove if you wish down to the crack, and go an inch behind and make a V-shaped groove, or make it in the form of a circle. It makes no difference. Now, thin it—unless you thin it very thin, I think it is better to cut down to the coronary band and the fleshy leaves, and pry it off. You get down to the surface of the pododerm in this way, whereas if you thin it you hardly ever get the horn thin enough. Take a knife and cut right through the coronary band and cut right out. The coronary band has great power of regeneration. In cases of quittor you can have two and a half inches of coronary band come away or slough, and it will grow from either end and unite, and produce a good coronary band that will send down a good shell of horn. Instead of fooling with the old scar tissue band, go to one side and cut through the band, even until you expose the bleeding point. Convert it into a flesh wound, and put a bichloride dressing on it. It will fill in with granulations, and make another solid band, and then you can allow the horn to come down. That is better than firing. It is the quickest way out. Just circumscribe that portion, strip it down, cut through the band, and cut out the scar tissue and treat it like a fresh wound. It will grow rapidly, and it will send down a solid sheet of horn.

There is another thing that is feasible or advisable. After you have finished and are ready to dress the foot, it is well to so dress it that the weight shall be shifted onto the opposite side. If we are careless we may have the foot so dressed that the weight is shifted onto the side of operation, so that there is tension and a tendency to open up and expand. But we can shift the weight slightly to the other side of the foot, because the joint is an imperfectly hinged joint and permits of a little lateral motion. Suppose the crack is in the inner or outer quarter. We will say it is on the inner quarter, and we have

done the operation. I would lower the outside and spare the inside if we have sufficient foot so that the foot shall strike first on the inner side, and the weight shall be shifted to the outer side. The expansion of this quarter is increased and the expansion of the inner quarter is slightly diminished. Suppose the foot is short, so that we cannot take any horn away. Then we will put on a shoe with a thin outer branch and a normally thick inner branch, so that when the foot comes to the ground it will come first with the thickened inner branch, and the inside will be relieved of expansive force and of spreading. We can put inner and outer heel calks on, but the outer should be a little shorter than the inner, so that it tilts to the inside and the weight is borne more largely on the inside. If you are sore on the inside of the foot, you walk on the outside, and vice versa. A horse has not got sense enough to do that, but you can arrange it so that he will have to do it by having the affected side a little long, so that it shall strike the ground and the weight will settle onto the opposite side of the foot. That is a practicable thing. In treating cracks of the inner quarter, leave the inside long, and if it is too short you can shorten the foot, and thicken the inner branch of your shoe so that it will hit the ground, and the weight will fall on the opposite side of the foot.

Q. Is it necessary to cut to the end of the crack?

Dr. Adams: No, it is not. If you find that your old scar is only in the coronary band, you do not need to cut below that scar in the coronary band at all.

Q. Do you shoe with a bar shoe?

Dr. Adams: If you have a complete crack it is well to shoe with a bar shoe. If the crack only passes down part way you can shoe with an open shoe. The bar shoe is the best shoe for the purpose, whenever we want to relieve a certain part from pressure, we can thereby throw the extra pressure on the bar. Suppose we use a bar shoe and give the animal good frog pressure, what will occur when the frog presses on the shoe? We will have a lateral expansion of the plantar cushion, a pressing outward of the lateral cartilage, carrying the coronary band, and the tendency would be to open the coronet. If we want to have the coronary band at rest in the region of the quarters, we ought not to have frog pressure. The thing that will press the coronet out is frog pressure, but we want the coronet to be relatively quiet until that coronary band fills in, and the ends grow together, so that it will be better not to have frog pressure on a rubber pad, which will produce it. So an open shoe would be better, no lateral expansion of the lateral cartilage, or an opening up of the cracks.

Q. Do you take the weight off of part of the wall back of the crack?

Dr. Adams: We always do whenever the crack is complete.

Q. A little later that crack will be down to the surface of the foot.

Dr. Adams: This crack which follows the old crack in the coronary band?

Q. It grows down, doesn't it?

Dr. Adams: Yes, is grows down with the horn.

Q. And later it will be—

Dr. Adams: It will be down near the bottom, and will be a bad crack; if it presses on the shoe, then it ought to be cut away and a plate put on. In this region we would put on a plate made of thin sheet metal with holes bored into it, and ordinary wood screws, three-sixteenths of an inch long, screwed directly into the wall, the plate bent so that it has just the curve of the quarter and at a red heat, pressed against the foot, until it makes a bed for itself, so that its surface is level with the surface of the foot, and then a little brad put into the surface, about each hole, and those little screws screwed into the plate, and that plate will not drop off. It is better than any strap around the foot would be.

Q. How about a horse nail driven through?

Dr. Adams: A good blacksmith can so bend his point that he can bring his nail through, but even when well nailed, it does not have the holding power that the plate has. I very much prefer the plate. We always put them on in our shop, a plate such as I have indicated, with three or four holes.

A member: It is mighty hard to get an ordinary blacksmith to do that.

Dr. Adams: The thing to do is to go to a hardware store and buy ordinary wood screws, three-sixteenths of an inch long. He can take a little sheet iron and bend it and let it burn a little bed for itself, and he can start his screws and he can use a screw driver. That is all there is to it.

If the crack does not go to the bottom you can put the plate on, and then imagine the crack carried to the bottom, from the point where it would strike the plantar border to the buttress may be shortened enough so that it will not touch the shoe, so that that quarter shall hang in the air, as it were.

Q. Do you use the Berlin plan?

Dr. Adams: Yes, but it is not a good device. The only place you can use it with safety is on the toe, and if you use hoof ointment it loosens the hooks; they get loose, and in the

melting snows of springtime they may drop out. The Vachette hook, while it looks all right, yet it lies on the surface, and the results are not nearly as good as with the plate. At the toe, where there is considerable convexity to bring the nail out where the wall has its greatest thickness, is where the horseshoe nail is more efficient, and when well placed it is a good method.

Q. Will you say something about contracted foot?

Dr. Adams: There is one sort of contraction that you men want to bear in mind as you get it in these particular horses, and that is contraction of wide feet. It is a peculiar contraction of the quarters that occurs in Percherons and Belgians and Clydesdales, horses that have only been shod a year or more, known as contraction of wide feet. It is not a contraction of a sole, it is not the ordinary contraction of one quarter or both, but it is what takes place between the coronary band and the horn, what you might call coronary contraction. A straight edge placed against this foot, just about touches from the coronet down to the plantar border. It sometimes happens in a wide foot that there is some groove in the quarters, very pronounced back at the buttress, which becomes shallow and disappears, as if a tight spring had been put around the foot at that point and driven into the horn, so that the horn is not straight from the coronet to the plantar border, but is constricted as by a corset. That is known as the contraction of wide feet, because it is seen in and around the feet of draft animals. It is seen more in that type of foot, and it is noticed here. When the straight edge is put against the foot, and the edge will not touch, it is as if a tight spring had been drawn around and kinked in the quarters—the animal affected is often quite sore. You will not see that the quarter is flattened. It has sensitiveness and the light tapping of a hammer is painful. There is a backing up of the blood in the digital and plantar arteries.

How shall we shoe him? How shall we shoe him to make that wall come out straight again? As long as the quarter presses on the shoe, that constriction will remain. It is better to put on a bar shoe with plenty of frog pressure, and no pressure on the quarter beneath this groove in the quarter that goes up to the last nail, and the foot is bent, so that it is under pressure from the last nail clear around as far as the branches of the foot forward. Good frog pressure; no quarter pressure at all, and pressure from the last nail here, clear back to the last nail on the other side. The foot should be kept fairly soft and kept greased. In three or four months when the horn at the deepest portion has grown down to the plantar border, the wall will be straight again and the compression of the fleshy

leaves at the bottom of that groove will be passed away. His blood will be no longer backed up in his arteries, and the animal will be sound.

That is the peculiar form known as contraction of wide feet, which is peculiar to the round feet of draft animals, and is often overlooked by the blacksmith.

Q. Will not that condition frequently result in a corn?

Dr. Adams: We would not call it a corn, but it is usually accompanied by some bruising of the fleshy leaves beneath the deepest part of this concavity. There is where the trouble and the inflammation is, in the fleshy leaves right along beneath the portion of that groove. And therefore we take the pressure off below. The more pressure below the more the bottom slides, and the deeper the groove becomes. In the ordinary foot about what fraction of an inch will the horn grow down in four weeks? It varies a good deal with use, food and age, but you might estimate that at a third of an inch. It may take three months for a one-inch growth. Some hoofs grow half an inch a month, but some will not grow more than a sixth of an inch. Exercise hastens it. Anything like blisters, exercise, green food and warm water hastens the growth. Cold weather will contract the blood vessels. Dry food, irregular exercise and much standing in the stable will, of course, slow the growth. The elasticity of the foot by softening and expanding the foot hastens it and invites the blood supply, but contracted foot by rendering the foot aenemic slows the growth.

HEMORRHAGIC SEPTICEMIA OF CATTLE

The Disease and Results of Serum Immunization

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This disease occurs usually in the acute form, less often in the subacute. It may appear sporadically or enzootically. The summer season marks the period of sporadic outbreaks, the fall and spring, the periods of enzootics. It is not unknown during the winter season, some of the most serious outbreaks with which we have contended occurring in December. A marked febrile reaction usually marks the onset of the disease, while approaching the termination, the temperature is apt to be near normal, and in a few cases observed even subnormal. Severe gastro-enteritis, necrotic pneumonia and an oedematous condition of the skin usually accompany the febrile symptoms. The disease is recognized in two forms, the pectoral and edematous.

The causative factor is the B. bovisepticus, one of the organisms of the septicemia or pasteurella group. It is a short, non-motile organism which stains heavily at the poles but only slightly in the middle, hence the descriptive term bipolar. This peculiar staining property is of greatest value in diagnosis by bacteriological methods. The virulence of the organism varies greatly in different outbreaks. Certain strains may kill rabbits in doses of 1-10,000 loopful or less while others may require several cubic centimeters. For calves and cattle the variation in virulence is even greater. One strain with which we worked killed a young calf in dosage of one loopful of agar culture while the other strains have been inoculated into calves of similar age in doses as high as 50 cc. of broth culture without fatal effect. The organism is readily destroyed by disinfectants, exposure for 1 minute to 1-5,000 bichloride, 6 hours to 3% phenol and 10 minutes to moist heat at 80° c. being sufficient. Dried virulent blood loses its power to infect after 14 days. (Kitt) Natural infection is thought most often to occur through the digestive tract, less often through injuries to the nasal or buccal mucous membranes. Pulmonary infection of rabbits is possible but doubtful for cattle. The finding of organisms in the soil in areas of outbreak points strongly to infections occurring through soil, especially in sporadic and beginning enzootic outbreaks. An interesting fact brought out in the course of our observations is that many of the cases originate among cattle feeding on cornstalks in fields newly broken from meadow. The virus shows an increased virulence under favorable conditions due to unknown influences. This

heightened virulence is retained for a time in the bodies of the animals, is gradually lost through attenuation and the enzootics subside. The symptoms of the disease vary according to the form but in the beginning are usually quite similar in the different forms. A rapid rise in body temperature to 104° F. or higher (107°) is accompanied by a rapid pulse, rough coat, trembling of the muscles, loss of appetite, and cessation of rumination. Constipation due to high temperature and loss of peristalsis is frequently observed at this stage, this later to be followed by symptoms of colic and the voiding of feces flaked with mucus and blood.

The pectoral form is marked by an acute pleuro-pneumonia. The thoracic cavity contains a sero-fibrinous exudate. The pleura is hemorrhagic and inflamed and may be covered with fibrinous membranes. The lungs show marked hepatization and the interlobular connective tissue is thickened and yellowish-white, due to the serous infiltration. This condition gives the lungs a mottled marbled appearance resembling that of contagious pleuro-pneumonia. The mediastinal connective tissue is gelatinously infiltrated and hemorrhages are numerous. Respiration is accelerated and labored, dullness occurs over different areas. A frothy serous discharge occurs from the nose and the animal stands with back arched and fore legs spread apart. This form of the disease frequently assumes a chronic type and the animal may linger for weeks, succumbing from exhaustion.

The oedematous form is characterized by swellings of the head and neck in the region of the throat and in the anterior sternal region. This is the result of an inflammatory oedema of the subcutaneous connective tissue. Incision of the swellings discloses a gelatinous infiltration. Enlargement of the joints, particularly those of the stifle and hock, frequently occurs, the gelatinous fluid from which will often contain the organisms in goodly numbers. The thymus shows marked petechiation, particularly in the anterior mediastinal region. The connective tissue of the pharynx and larynx shows marked gelatinous infiltration, while the mucous membranes of both are highly inflamed and thickened and dotted with hemorrhages. Difficulty of deglutition results in the accumulation of saliva which dribbles from the mouth in frothy, tenacious strings. Difficulty of respiration increases and the animal dies of asphyxiation. Yellowish or reddish serous fluid is often found in the abdominal cavity in considerable quantities. The rumen in rare cases shows acute hemorrhagic inflammation, while in the small intestines this condition is common. The large intestine usually shows less of such changes, but in nearly all cases the rectum is highly inflamed and contains much

slimy, bloody exudate which covers the feces. The wall of the gall bladder and the tissues surrounding are infiltrated with a gelatinous fluid and the bile is often thickened and yellowish-red. In most cases the spleen appears normal, but occasionally is soft and swollen, while the capsule is covered with fibrinous inflamed membrane.

Either of the above forms may be accompanied by nervous symptoms. The animal behaves in an insane manner, traveling in a circle, bellowing and struggling blindly into any obstruction which it may encounter. It is not unusual for such animals to attack an observer, rushing madly at him.

In holding post mortems for diagnosis we are accustomed to look for the following changes: those of the serous and mucous membranes. These showed marked hyperemia and hemorrhages. The subcutaneous connective tissues of the head, throat and neck are infiltrated with a straw-colored, sometimes sanguinous, gelatinous fluid and usually show well-marked hemorrhages. The thymus gland in the anterior mediastinum is usually dotted with hemorrhages varying in size from minute petechiae to a size of 4 or 5 millimeters. The infiltration may extend to the deeper layers of the muscles, and is marked in the connective tissue of the larynx and pharynx. The epiglottis is petechiated and the nasal mucous membranes are highly inflamed. The lymph glands, particularly those of the head and neck and the peri-bronchial and mediastinal, are acutely swollen, inflamed and juicy. The intestines show a markedly acute inflammation and their contents are usually thin and grayish yellow in color. In most cases the spleen shows no changes, in a few it is enlarged and highly inflamed. The liver is usually normal in acute cases, but in chronic shows cloudy swelling. The gall bladder is distended and the bile gelatinous and ropy, and from it the organism may be isolated. The pericardium, epicardium and the heart muscle are studded with hemorrhages, and particularly in chronic cases the pericardium contains a serous exudate. The lungs, in the pectoral form, become hepatized, are dark red or brown and show a mottled appearance on the cut surface due to the serous infiltration of the interlobular connective tissue.

The following is a preliminary report of an effort made in the Research Department of the Veterinary Division of the Iowa State College to produce a serum for the treatment of hemorrhagic septicemia:

Literature, American or foreign, records little information concerning the treatment of hemorrhagic septicemia with serum. That which has been tried has been principally in the treatment of the disease in sheep, called by Meissner and

Schern "Septicemia pluriformis ovium." (1) These investigators, in 1910, established the etiological relationship of bipolar bacilli in the disease which for a long time had been variously called catarrhal fever, glanders and gangrenous rhinitis of sheep. By inoculation of susceptible animals they succeeded in reproducing the disease, both in its acute and sub-acute form. Subsequent efforts to produce a potent serum for the treatment of the disease were attended with success and the results of their efforts towards serum immunization are given later.

The serum used was prepared by systematic immunization of a horse over a period of nine months, beginning with 1 loopful of culture of hemorrhagic septicemia organism and increasing the dosage to a limit of 4 agar cultures of the same. Three cubic centimeters of this horse serum protected rabbits against 1-1,000 of a loopful of culture, the control animals dying.

In the treatment of sheep the investigators recommend the serum as a prophylactic agent, repeating the treatment in four weeks. For those animals very sick or suffering with the chronic form they found the treatment not highly satisfactory, since the majority died. Those slightly sick or apparently healthy responded to treatment. In this latter class, in addition to the serum, 4 cc. of an extract of the organism were simultaneously administered, and four to six weeks later the same quantity of extract alone was given. This resulted in the production of a fairly permanent immunity. The dose of serum was 10 cc. for the apparently healthy and 20 cc. for those sick. The serum treatment is to be accompanied by rigorous hygienic measures such as thorough disinfection of the stables, cremation of the dung and thorough drying of the bedding, etc. Their conclusions are as follows: That the serum possesses both prophylactic and curative properties, but that good results were not obtained in all cases; that a number of outbreaks were treated in which the serum lacked both the properties to protect and to cure. (2) Raebiger, in 1913, following the same method of Schern and Meissner, reports even better results obtained with the serum than were had by the latter, while (3) Raebiger, Spiegel and Schern, in 1915, had further opportunity of testing the serum. In this latter case an immune serum was produced by systematically immunizing sheep with repeated and increasing doses of B. ovisepticus. The serum was tested upon white mice in doses of .2-.3 cc. intraperitoneally against .1 cc. of a virulent strain of B. ovisepticus given subcutaneously. Mice thus treated

(1) Archiv fur wissen und prakt. Tierheilk. Bd. 36.
(2) Deut Tierartz. Woch. Mar. 8, 1913.
(3) Deut Tierartz. Woch. Nov. 20, 1915.

were immunized while control mice died within 3 days. The following tabulated results show the effect of the treatment:

No. dead before treated	No. apparently healthy	No. very sick	No. slightly sick
328	2,764	112	138

Of these, twelve of the apparently healthy died subsequently to treatment, 30 or 22% of the slightly sick died, and 53 or 47% of the very sick died.

However, the percentage mortality (9%) of those dying before treatment was reduced after treatment to 3.7%. The quantity of serum used in the treatment of the various classes was 5 cc., 10 cc. and 15 cc.

Encouraged by the results thus obtained in the treatment of sheep, Dr. Schern upon his arrival at the Iowa State College in 1914, began some experiments to determine the value of serum treatment of bovine hemorrhagic septicemia. Work was carried on in a small way until the summer of 1914 when Dr. Schern returned to Germany where he has since been detained. The author then, in additon to his regular work, took up the problem and has pursued it as time permitted until the present.

The first difficulty encountered was the securing of a suitable strain of organism for the work. Many efforts were made without success. In September, 1914, we were rewarded by securing a culture from a cow belonging to the college farm. This animal, about two weeks after calving, became ill and was brought to the clinic with a clinical diagnosis of hemorrhagic septicemia. Inoculations of blood made directly from the cow before death into six rabbits proved negative. After death, an extract from one of the cervical lymph glands was injected into a rabbit, and proved fatal in twenty-four hours. One cc. of twenty-four hour bouillon culture from this rabbit killed a second in seven hours, but 15 cc. of bouillon culture failed to kill a calf when injected intravenously.

By several passages of this culture through rabbits and English sparrows it seemed to gain in virulence, for, on February 12, 1915, an inoculation of 10 cc. intravenously into a calf six weeks old resulted in the animal's dying in four hours. Post-mortem findings of this calf were positive and cultures were obtained from the heart blood and from extract of the lymph glands. Subsequent tests of the culture were made in the same and higher dosage upon calves of different ages with positive results. A second highly virulent strain was later isolated from a dairy calf. One loopful of agar culture of this strain injected intravenously caused the death of a three-weeks-old calf within twelve days. This is the smallest fatal dose thus far obtained.

The first animal hyperimmunized was a small calf which had been brought to the clinic several months before, suffering a mild but chronic form of septicemia. This animal had apparently recovered although it was undersized and unthrifty. Immunization was begun by giving it first 2 cc. of blood from the cow that furnished the strain of organism. Then followed 6 cc. of blood from a rabbit killed by intravenous injection of the culture. Later this animal received a number of injections of bouillon cultures of the organism amounting in all to not over 100 c.c. On December 5, 1915, the calf was killed and its serum preserved. This was tested on rabbits with the following results:

Date	*Rabbit*	*Size*	*Culture*	*Serum*	*Results*
12-9-14	4	2,250 gm.	1-10,000 loopful	1 cc.	Died Dec. 10, 1914.
	5	2,000 "	"	2 cc.	Lived.
	6	2,050 "	"	3 cc.	Lived.
	7	2,150 "	"	4 cc.	Lived.
	8	2,150 "	"	5 cc.	Died Dec. 16. Abscess due to subcut. inoculation.
	9	2,350 "	"	5 cc.	Lived.
	10	2,000 "	"	0 cc.	Died Dec. 10.

On January 18, 1915, rabbits Nos. 5 and 6 were given 1-2,000 of an agar culture of the same strain of organism without showing any ill effect, while control rabbits with the same dosage died within twenty-four hours.

Rabbit No. 7 was pregnant when put in the test and after eight days gave birth to three young. One of these was injected March 9th with 1-10,000 agar culture of the organism without effect, while a control rabbit of about 200 gms. greater size died within twenty-four hours. This is mentioned only as an interesting side light on the main question. Of the other rabbits used in this test all lived until they were killed in the course of the regular laboratory work, the last one about fourteen months later. Opportunity for testing the serum of this calf in the field occurred only once. This was in an outbreak on a farm west of Gilbert. The owner had four calves of probably 300 pounds weight, which he was fattening for market. In April all four calves became ill at about the same time. One of them died one day and two more the following. The local veterinarian was called and held a post mortem upon the last two dead. Lesions of hemorrhagic septicemia were found and he gave the owner no encouragement concerning the fourth calf which showed a high temperature, was depressed, manifested dyspnoea and was discharging at the nostrils. I was informed concerning the case and went to the farm in the afternoon prepared to hold a post mortem and secure some clinical material and possibly a new strain of organism. I found the animal in better condition than I had expected and

instead of killing it, I gave 100 cc. of the immune serum subcutaneously. The calf recovered and several months afterward showed no ill effects of the disease.

The second animal to be hyperimunized was a sorrel mare of 950 pounds weight. The immunizing process is given in detail for this animal to indicate the care that is necessary in the administration of the cultures in order to avoid serious results. The mare had received 1 loopful of live culture of a strain of B. bovisepticus subcutaneously June 1, 1914, 3 loopfuls of the same on June 15th and ½ agar culture June 25th. Nothing more was done with her until November 23d when we began hyperimmunizing her by giving 2 cc. of a twenty-four hour bouillon culture attenuated for 1 hour at 58° c. This culture was the strain secured from the cow previously mentioned and reference will be made to it subsequently as the "farm" strain. This strain had had several animal passages up to this time, including rabbits, sparrows and mice, the last one being through a mouse 2 days previously. The tabulated treatment of the animal is given below:

Date	Culture	How secured	Strain	Quantity	How given
11-23-14	Bouillon	Mouse	Farm	2 cc. heated 1 hr. at 58 degrees	Subcut. Subcut.
11-30-14	Bouillon	Mouse	Farm	1 loopful of above in 4 cc. NaCl	Subcut. (a)
12- 8-14	Agar	Rabbit	Farm	½ agar in 4 cc. NaCl	
12-15-14	Agar, 48 hr. old	Rabbit	Farm	6 loopfuls in 10 cc. NaCl	Subcut.
12-22-14	Bouillon, 24 hr. old	Rabbit	Farm	20 cc.	Subcut.
1- 4-15	Bouillon, 96 hr. old	Rabbit	Farm	10 cc.	Intrav. (b)
1-13-15	Agar, 24 hr. old	Rabbit	Farm	9 agar in 50 cc. NaCl	Subcut. (c)
1-25-15	Bouillon	Calf No. 6 (d)	Farm	20 cc.	Intrav. (e)
2- 2-15	Bouillon	Calf No. 6	Farm	70 cc. of 14 day old cult.	Subcut.
2-17-15	Bouillon	Calf No. 7	Farm	30 cc.	Intrav.
2-22-15	Bouillon, 24 hr. old	Rabbit	Farm	50 cc.	Intrav.
3-16-15	Bouillon, 24 hr. old	Rabbit	Farm	100 cc.	Subcut.
3-29-15	Bouillon, 24 hr. old	Rabbit	Farm Curtis Cole Sheep	90 cc.	Intrav.
3-30-15	Horse died				

(a) Animal very sick. Lost 5 pounds weight.
(b) Rapid, labored breathing.
(c) Swelling of stifle joint, lame. The culture used this time killed mice in .3 cc. dose subcutaneous injection in 6 hrs.
(d) Calf killed by 10 cc. intrav. injection.
(e) Animal badly depressed.

The loss of this animal indicates that the immunizing process was too rapid, and that increase of dosage was far beyond the point of safety. It will also be noted that the last injection contained in addition to the Farm strain of organism three other strains recently isolated. This may in part account for the fatal results. We have profited by these mistakes and the later animals immunized have not been crowded. We have assumed as a safe limit for increase in dosage not over 3 cc.

February 1st, 1 liter of blood was drawn from the mare's jugular and tested on rabbits. It did not show potency sufficient to protect against 1-1,000 of a bouillon culture, but was used successfully later in the treatment of six head of calves in the field.

At present we have four animals, two horses and two calves, which have been treated for different lengths of time, some of which are yielding serum that is giving satisfactory results in field tests. One horse has been given a total of 355 cc. of bouillon cultures in a period of six months, and at the last injection received 50 cc. Her serum has been used successfully on 38 head in the field. The two immune calves have each received 500 cc. of bouillon culture in a period of nine months. Field trials have been made with this serum on 34 head.

The serum of these calves was not potent after 2½ months' treatment, totaling 50 cc. bouillon culture, but after 5½ months' treatment totaling 200 cc. bouillon culture it was used successfully in 10 head (Cline, McDonald, Lowden) in the field. The serum from the horse was tested first after 4 months' immunization using a total of 225 cc. bouillon culture upon 3 head of sick calves (Morris), two of which died.

Our purpose in carrying out the experiments as suggested above may be stated as follows: There seems to us little opportunity to determine accurately the prophylactic value of the serum. Our experience is that comparatively few animals of a herd give evidence of infection, such being usually confined to 5 or 6. The infection seems to be self limiting for some reason, possibly due to the erratic behavior of the organism which will show a remarkable activity and virulence for a time and then suddenly becomes inactive. This being the case, serum used on a healthy herd or on one in which the disease may be present but in which the mortality will be low without treatment, will seem to show remarkable results when the percentage mortality is computed. Such results are in no way to be credited to the serum. It may be argued that by subsequent treatment of supposedly passively immunized animals with a virulent strain of organism it can be conclusively proven whether or not actual immunity has been conferred. Such treatment, in our experience, is highly unsatisfactory and unreliable because of the difficulty of conferring the disease by inoculation with cultures. In several instances we have not been able to kill young untreated calves by inoculating intravenously with as much as 50 cc. of bouillon culture, nor have we in some of these cases been able to show any but a temporary reaction even to so large a dosage, subsequent behavior of the animal showing that but little if any lasting bad effect was felt. True, we have shown that rabbits treated with serum alone or with serum and virulent culture simultaneously, acquire immunity that protects them against doses of the organism that readily and quickly kill untreated rabbits. But we find a very constant and uniform virulence of our cul-

tures for rabbits, hence tests upon them are very satisfactory. Reasoning then, from successful treatment of laboratory animals, we might conclude the same thing would be true of cattle, but at present at least, I cannot see that we have any right to draw such conclusions.

Since then we cannot satisfactorily determine the value of serum as a preventive agent, our purpose has been to demonstrate its value when used upon animals already showing symptoms of infection. Of those cases treated we have had positive clinical and post-mortem diagnosis of the disease in animals dead from the same herd in every case. We have had the same plus positive laboratory diagnosis for all except five animals. We feel we know then that the animals treated were actually suffering from hemorrhagic septicemia. The results obtained on the comparatively few animals treated, which gives a value of 89.74 per cent success, encourage us to further efforts, and we hope in time to present further and more complete data upon the subject.

Below is appended a tabulation of the cases treated and the results obtained up to the time of the preparation of this article:

Owner or veterinarian, location.	Number in herd.	Number sick.	Number healthy.	Number died before.	Treated.	Number sick recovered.	Number sick died.	Number healthy O. K.	Number healthy died.
Dr. Nelson, Gilbert	4	1	0	3	1	1	0	0	0
McKone, Napier	8	2	4	2	6	2	0	4	0
Cline, Clarion	7	4	0	3	4	4	0	0	0
Morris, Ames	9	3	2	4	3	1	2	0	0
McDonald, Ames	3	0	2	1	2	0	0	2	0
Lowden, Winterset	12	7	0	5	7	7	0	0	0
Morrisey, Winterset	44	17	27	1	17	17	0	27	0
Bly, Radcliffe	14	11	1	2	12	8	3	1	0
Lavender, Clarion	14	3	9	2	12	3	0	9	0
Chapman, Truro	5	4	0	1	4	3	1	.	.
Macklin, Coon Rapids	84	10	72	2	10	10	0	72	0
Truax, Sac City	19	2	14	3	2	2	0	14	0
Bolton (I. S. C.), Ames	18	8	10	0	18	8	0	10	0
Nelson (Dairy) Farm, Ames	11	3	7	1	10	2	1	7	0
Luithly, Richland	7	2	4	1	2	1	1	4	0
Totals	259	77	163	31	110	69	8	150	0

Percentage of recovery of sick that were treated, 89.74, September 5, 1916.

Percentage of mortality before treatment, 12.

Percentage of mortality after treatment, 3.5.

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"PROBLEMS OF THE PROFESSION"

DR. D. S. WHITE, OHIO STATE UNIVERSITY

Mr. Chairman and Gentlemen: I will not dignify what I have to say this evening with the term "address." I am not going to call it a speech. Neither will I speak of it as a talk. It will be, I fear, a mere chat.

One of the fundamental principles to which I adhere very strictly is never to attempt an extemporaneous effort without due preparation. A great many of you here who missed me, if you did miss me this afternoon, may have thought that I was working to get together or to knock together this chat. As a matter of fact, I was asleep at the hotel. My partner, Dr. Adams, and I paired off today. If I would keep away while he was talking, he would keep away while I was talking. So he is sleeping at the hotel now. (Laughter.)

I really hardly know how to approach this subject. I feel somewhat as one of our judges in Columbus did. He is a rather ready speaker. He has spoken on the stump and he has addressed many an audience in the last thirty years in the state of Ohio. In fact, he gained such a reputation as an offhand speaker that they invited him to address the convicts one Sunday in the state penitentiary. He arose before that audience—the audience was large because the attendance was compulsory—and he seemed to be appalled at the sight that he saw there and the faces upturned to him, and he looked at them a little while, and he said, "Boys, I am so glad to see so many of you here today." And that was as far as he could get with his speech. He had to sit down.

I am not quite as given to inspiration as a colored brother I heard of once. It seems that the African division of the Methodist Episcopal Church was having a convention in the city of Washington. Among the delegates were two preachers, colored, one an educated man and a graduate of Oberlin College. The other was an uneducated darkey. They were walking arm in arm down Pennsylvania avenue, and they got to discussing how they prepared their sermons. The uneducated brother said to the other, "Reverend, how do you prepare your sermons?" "Well," he said, "I don't do much at it until about Wednesday. Then I think about a subject that I would like to preach on. And I go down to the library and I read all the books that I can get hold of containing anything applying to that subject. And then I go home and look over my Bible, and I pick out an appropriate text. Then I write this all out on my typewriter, and when Sunday comes I get

up before my congregation and deliver my sermon." "That ain't no way to preach the gospel." "Well," he said, "how do you do it?" "Well," he said, "I depends upon inspiration." "I would be afraid to do that," said the other. "I would be afraid sometimes that the inspiration would not come." "No," he said, "it always comes to the man that's got the right heart. Now," he says, "I can preach without any preparation at all on any text. I can preach on anything; I can preach on even a single word." Well, the educated darkey thought to try him out, and he said, "I will give you a word and see if you can preach a short sermon on it." He said, "All right. Try me out. Give me a word." "Well," said his brother, "you preach a short sermon on the word 'pill.'" That seemed to stump him for a moment, but finally he said, "I got it. I'll get up before my congregation and I'll say, 'Brethren and sisters, for the rheumatics they is certain pills; for the lung fever they is other pills; for the neuralgy they am pills; for all the ills of the flesh, brethren and sisters, there is some sort of pills; but for the ills of the soul they is but one pill, and that am the Gospil.'" (Laughter.) That was not a bad sermon.

But I haven't got the inspiration. As the gentleman who introduced me said, I do not know that he had me in mind as a matter of fact when he made those complimentary statements. In the last quarter of a century it has been my opportunity to contribute somewhat toward the progress of the veterinary profession. When I was a student of veterinary medicine, from 1888 to 1890, I think I spent only two years on the veterinary curriculum, that was then offered at the Ohio State University. I know I took the course backwards. I remember getting my anatomy as a senior. But the amount of anatomy we got there at that time was so small that it was pretty innocuous and homeopathic and attenuated so that it did not make very much difference. The whole curriculum was given by one man, old Dr. Detmer. He was a battle-scarred hero, who came to this country from Germany. He taught veterinary medicine at this institution, at the University of Illinois, and at the Ohio State University for many years. The old man was very far ahead of his time. He was many, many years—I was going to say decades, ahead of his time, as far as some of the fundamental sciences underlying veterinary medicine are concerned. He was one of the first men in Ohio to do anything in bacteriology, and for many years he taught the only bacteriology which was taught in the state of Ohio. I will admit that he was interested only in certain phases of bacteriology, particularly the photographing of bacteria, and he left behind him a great many very beautiful photographs which he had taken. He was also one

of the best known microscopists in the United States, and was at one time the president of the American Microscopical Society. I remember he had a special microscope built for himself, almost as big as a twine binder, and a great deal more elaborate. No one was allowed to go anywhere near that instrument. Even his wife was not allowed to dust it, or perhaps even to go into the room where it was. He could not adapt himself very well to conditions. He was his own worst enemy, perhaps, and while he failed to accomplish what he set out to accomplish in the way of establishing a veterinary school at the Ohio State University, nevertheless he did inspire a great many people, and he gave me what little inspiration I received in that direction. He left the university many years ago under a cloud, as it were, and his last hours were spent in sadness. He did not at any rate reap the reward which should have come to him. He was a man who was his own worst enemy.

Veterinary medicine, of course, is like everything else. It has had to evolve, and the processes of evolution are very slow. When our ancestors landed in this country in New England, and attempted to capture the forests and wring from the poor, rocky soil of that region a living, surrounded as they were by the Indians, wild game and many things which affected their well-being and prosperity and made life hard and arduous, obviously the man who was actually a hewer of wood and a drawer of water was the only man who could be tolerated in such a community. Any man who could not go out and fell trees and who could not do the hard labor which was required in the pioneer days could not be tolerated, obviously, in the colony.

In those days, therefore, you found no one but actual workers, men who worked with their brawn rather than with their brains.

As the trees disappeared and the fields began to be tilled and the crops harvested, and the folk a little more prosperous, then they began to feel wealthy enough to support some individual in the community who was not an actual hewer of wood and drawer of water. They seemed to feel they needed some spiritual advice. You know man is a religious animal. There is a little religion in all of us, whether we admit it or not; and they wanted a preacher, and most of the colonists looked upon the preacher as a sort of a loafer. He slept later than the rest, and he wore a standing collar on Sunday and a roll-tail coat, and his hands were usually soft like the hands of a town lady. But he was tolerated because they seemed to feel that his spiritual guidance was necessary; and probably some of them looked upon him as a sort of a fire escape,

which would prevent them from getting into the clutches of the master of the inferno. I do not know about that.

And finally it became necessary—the first preachers they imported, but finally it became necessary to educate these preachers. So John Harvard started a little school in Cambridge to educate preachers to go out and preach the gospel to the colonists. And they got a little richer as times went on, these colonists did, and they felt, "Now, we ought to have somebody to take care of us when we are sick;" as it used to be like on the old ships the captain was the ship's doctor. He had his medicine chest, and he dished out or doled out what seemed to him to be indicated in each case that was presented to him, from the crew. So they thought that they ought to have a doctor, and for a while they combined these two, and that is a peculiar thing that runs through the past history of medicine, this dual position of the doctor and the preacher.

You know in the early times priests used to act as physicians and treat the body as well as the soul. So Harvard established a medical department, and very often in the community the preacher was the doctor and also the one who ministered to the spiritual needs of the community.

Well, in those early days the live stock did not amount to very much. There was not very much live stock. What there was of it ran wild in the woods. If you should go down into southern Alabama where I spent a couple of weeks a year ago last Christmas, you will see not a very fertile soil for the veterinarian. In fact, there was not any doctor in that community except a man who lived on an island a distance of twelve miles away, and he was just spending the winter there, and he was not a real doctor; that is, an ethical doctor. I think he made some patent medicine that he had made a fortune out of; but the people of that community were compelled to patronize him.

But to return to the live stock phase of it. Running in the woods all the year around were what few cattle they had, and what few goats. The soil was too poor to support sheep, so they compromised on goats. They had not in the town I was in any horses, but I did hear that there were two horses farther north in the county. None of those animals had any particular value. I presume ten or twelve dollars would be a good price for a cow in that particular part of Alabama. And I imagine in the early days things in New England, as far as live stock were concerned, were in about the same condition.

But as time wore on the people began feeling rich enough to leave the farm and come to town, and the cities began to

develop, and the farms became more fertile, in the sense that they cleared more land and could raise more crops, and the cities furnished them a market. Without going into the economic side of the situation you can picture in your own mind what happened.

And then we began to import live stock from the old country. Ohio, my native state, went into the live stock business rather heavily. Among the first Short-horn cattle imported to the United States came to Ohio, and the first swine, Berkshires, from which emanated finally the Poland China breed, I believe it was, was imported from abroad into Ohio, and incidentally it seemed to have brought over with it the hog cholera, and that has made business and occupations for a great many people. If it were not for the hog cholera I do not know what a great many of us would do.

That development in Ohio came because the early settlers of Ohio came from New England, and they settled in Ohio in the roughest and hilliest part of the state, for two reasons. First, because it looked more like back home to them, perhaps, but the second and more tangible reason was that in the glacial part of the state it was largely a swampy, marshy country, and they had the ague and the shakes, and they shook so hard that they could not run a plow and till the fields.

So, without tracing the present development of this country any further along these lines, you can see that as we got better live stock and as the value of the live stock increased and the number of domesticated animals increased, it became necessary that some expert service be rendered. Now, experts in a country like America were very easy to get. They were omnipresent. So it was not long before each community not only supported a minister of the gospel and a physician, but also a horse doctor, or a cow leech, as some of them were called. Many of them were born in the manger trough, so to speak. Their fathers, perhaps, had tinkered at it a little, and they handed down from family to family certain traditions and certain cures. I have in my possession some old books which were presented to me by an old gentleman who was a non-graduate veterinary practitioner for about sixty years, whose father before him was also. It is very interesting to read those books. They are mostly made up of prescriptions and names, because in those days about all they wanted to know was what the name of the thing was so they could look in a book and get the prescription and give it. That was a simple process.

These men served their time, perhaps. The conditions of the country would not warrant any more than that.

And finally in some of the larger centers there came from

abroad, veterinarians. Usually in each state there would be a graduate of one of the English or one of the continental colleges wander over here. Some of them never really graduated, but they took their semesters or two years' work, and when they came over to this free and enlightened country, where we did not know much about England and continental conditions, they could pose as graduates. They knew more, at any rate, than anybody else in the community, and they served the people and received for their service probably about what it was worth.

Then we saw that we could not depend upon the charlatan who grew up among us, usually a man without much general education, a man with no training, technical or professional. We could not expect the old country to furnish all of the veterinarians, so it became necessary for us to start schools of veterinary medicine in this country. There were one or two feeble attempts which failed.

A school was started in Boston which ran for a time, I believe in early fifties, and then went under. Another one was started in Philadelphia. But one of the first schools upon this continent to be really successful in the sense that it did give some technical and professional training to its matriculates, was the old Ontario Veterinary College at Toronto, Canada. I have great respect for that old institution. I never saw it. I never met the man who founded it. None of my kith or kin ever graduated there or were in any way connected with that institution. Of course they had a short curriculum. The first curriculum consisted of five, perhaps six, months' training during the winter season; and then the students came back the next winter and took it all over again with one or two exceptions; but what he did get he got pretty well; and if you should look over the roster of the various state and community veterinary societies in this country, or the roster of our American Veterinary Medical Association, you will see that some of the leaders have emanated from that old school up in Toronto.

It furnished, with remarkably quick delivery, I will admit, what was needed in this country at that time, a large number of veterinarians, not thoroughly trained and scientifically educated veterinarians such as existed at that time on the continent of Europe at least, but men who knew more about veterinary medicine, men who were more skilful and dexterous in the applications which they had learned than anybody else in the community.

I remember thirty years ago going to the city of Cincinnati and visiting a medical school down there, not a veterinary school; and you never saw such a looking bunch of rummies

as sat before me on the seats, and I said to the dean of the school, "Do you expect to make physicians out of those fellows?" He said, "No, not at all, but each one of these men will know a little more about medicine than anybody in the community in which he goes to practice. He will stay in that community until he gets some clinical experience, and by that time he will probably have killed so many folks that he will have lost his reputation, and then he will move to another community; but having this experience back of him he will make good at the second or at least at the third stop." (Laughter.) That fellow was a philosopher. He charged those boys $100 a year, and he gave them about $100 worth of medical information, nothing more and nothing less. So it was a square deal all around.

All of the law schools and all of the medical schools with few exceptions, and all of the veterinary schools without any exception were, of course, in the hands of private individuals. They found that this institution up at Toronto was serving a dual purpose. It was not only furnishing veterinarians to the United States and the Dominion of Canada, but it was also making the owner of the school a wealthy man. I do not begrudge him a single cent that he got from the school, but he made a lot of money. He became a prosperous and an influential citizen of the Dominion of Canada, and he was one of the best known men in the dominion.

And they saw that that was a pretty good thing. So schools started in various parts of the United States, one of them in the city of New York, by a Frenchman by the name of Liautard. Dr. Smith was a Scotchman, and when you put a Frenchman and a Scotchman together in a race for the dollar, you always know which horse to play in the game. The Scot will have the dollar before the Frenchman gets started. But still he sort of copied after Dr. Smith's school in Toronto. Smith furnished the example, and he made a pretty good pattern after him.

It would take too long to discuss in detail the development of the private veterinary schools of this country. It is a passing phase, in my mind. It came in and marks an epoch in the evolution of our profession in this country.

Then some of the states began to become interested in veterinary medicine as a state project. At old Cornell University there was a great old man, old Dr. James Law, who came there away back, I think, in 1868; and when Ezra Cornell sent Dr. White, who was then, I believe, a trustee of Cornell University, to Europe to get a veterinarian, he left it entirely to him as to whom he should select; and he selected James Law, who came here. He was also a Scotchman and a

very scholarly, dignified, well-read, polished and well-educated man. He had indirectly, probably more than directly, a wonderful influence upon veterinary medicine. Law was in constant contact with some of the men who afterwards became great educators in this country. I do not care where I go—I visit a great many universities in the course of a year—I always find some old Cornell man who has been a professor at this institution perhaps for many years, and when he finds out who I am he asks me if I do not know Professor James Law; and this experience was repeated right on the main street of this town only yesterday. I do not remember the man's name, but someone introduced me to an elderly gentleman, and he said, "Oh, yes, you know Professor Law, of Cornell. I used to take lectures many years ago under him there."

And through Dr. Law there was spread over this country, as Cornell was a growing institution and sending abroad to the other educational institutions younger and smaller than it was, many men who made up their faculties, and these men spread the good word all over the country. They were not necessarily veterinarians, but they always spoke a good word for the veterinarians, and whenever anybody attempted to decry the profession they thought of old Dr. Law and they immediately sprang at him, figuratively speaking, and told him that he did not know what he was talking about.

And that was something that Professor Law should be given full credit for.

I have had a little the same experience at our own institution, if you will pardon me for being personal, but when they turned over to me the veterinaryship on our campus I found that she was a good deal like the Irishman's gun, which needed a new lock, stock and barrel. She was mastless and rudderless, and both sides were caved in. We had a bad reputation on the campus and off the campus. About a year ago I was very much pleased and somewhat impressed by the university faculty when I read to them the result of the meeting of our faculty, the veterinary faculty, which had declared itself unanimously for a four-year curriculum, and a full high school entrance requirement; the members of that faculty, many of them staid old gentlemen whom I had never seen out of a frock coat, began to cheer, and in that way they expressed their appreciation of the veterinary college, and at the same time—and this is the part that I was interested in—it told me that in twenty-five years we had developed on that campus and over that state an entirely different feeling toward veterinary medicine than had ever existed before.

And that is one of the first things in the progress of vet-

erinary medicine, or in any other profession that you have got to fight for, and that is recognition. Once you get that the rest comes easy. Why, I can remember how hard Dr. Detmer fought to get an appropriation out of the state legislature in 1890 to build a veterinary building. He got $4,000 for the building and equipment. Why, I would be ashamed to ask the legislature for $4,000 now. Surely a much larger amount would come readily if asked for.

Just a few remarks about some of the things needed to make our progress secure. The first thing I find on the back of this envelope (referring to an envelope on which the speaker had made memoranda) is the abolition of commercialism in veterinary education. We still have that spirit. Without going into unnecessary and irrelevant detail in this regard, suffice it for me to say that we have institutions of veterinary learning so-called, still upon this continent, which are more interested in the commercial than they are in the veterinary aspects of veterinary education. The dean of one of these institutions, and one of the most prosperous ones was in my office the other day trying to get me to assist him to apply the brakes in the American Veterinary Medical Association, so that the entrance requirements would not be raised at this time or in the near future, and he wanted to argue his case. I said, "Doctor, I cannot argue with you. We are not talking about the same thing. I am talking about veterinary education, and you are talking about making money." And those two things used to, perhaps, but today they have not anything to do with each other.

Most of you are practitioners, I presume, but it seems to me your efforts as practitioners should be directed in this regard. It is a common statement, it has become stereotyped, that what we need in this country is not so much more veterinarians, but veterinarians of a better quality. I am firmly convinced that this is true. I am firmly convinced that the greatest problem of all before veterinary educators is to induce men of broader and better training to come into the profession, men who will become leaders, men whom we now look upon as exceptional in our profession. We need more of them.

Now, this can only come by shutting the portals, the gates, of the profession a little closer than we have heretofore. As long as we leave it an open platform, as long as we permit anyone to enter our colleges, the men who have the qualifications demanded by medicine or by law will go into medicine and into law, because they do not want to waste, as they put it to themselves, their time in this acquirement in the way of preliminary education.

It used to be even worse in medicine. Dr. Eliot, of the Medical School at Harvard University, now emeritus president of Harvard, told me that when he taught chemistry in the Harvard Medical School he could not give written final examinations because so many of the students were unable to write the English language correctly enough to express themselves.

We should then increase our standard of information. Then we should intensify our curriculum. I think that is being done in most of the institutions, but I know in ours in all respects it is not true. By intensifying the curriculum I mean one that is in every regard professionalized.

We have at all of these institutions two groups of teachers, teachers of the laboratory subjects, the basic and fundamental subjects, and then the teachers of the clinical subjects. I am not making this application local at all. The teacher of the laboratory subjects is too often not a veterinarian. Our professor of chemistry, for instance, for many years failed to teach our students chemistry, not because he, the professor, did not know chemistry, but because he had absolutely no conception of the kind of chemistry, the applications of chemistry which our students needed. Our professor of zoology had not the faintest conception of the needs of the veterinary students in that regard. He came to me—I can tell this on him now because he is dead, and you can always tell the truth about the dead—he came to me and asked me if I would turn over the seniors in parasitology, I think they called it, to him. He said he would like to teach the veterinary students. I said, "What little parasitology is being given I am giving myself, but I will give you an hour a week, and you take those men and tell them all about the worms which infest domestic animals." I sent him over the entire senior class, two men. Six weeks later 50 per cent of the class came to me and said, "We do not seem to be getting very much out of this course." I said, "Why not, Mr. Jones?" "Well," he said, "we have been working over there six weeks on the earth worm. We have been making cross sections of the earth worm;" and the term which we had then was only about twelve weeks, and the term was half up; "and from the work the old professor has laid out I think it will take all the rest of this term before I get through with the earth worm." That professor found out that those boys had studied histology and that they could use the microtome, and that they could do things that many of his other students could not do, and he was interested in the study of the earth worm, and he put that work on them, and they worked at it, thinking that something better was coming, but something better never came. So that class grad-

uated without any knowledge of parasitology. That may seem an exaggerated case, but it is not exaggerated. It is a fact.

Now, another thing we need is more good veterinary teachers. I made the statement in Detroit the other day that I did not believe there were enough men, veterinarians, who were trained pedagogues in the United States to man one veterinary college, provided the college was to be adequately manned. I doubt if this statement anywhere near approaches the truth, that if at any one of the veterinary schools there are enough qualified pedagogues. Veterinary pedagogy is a profession in itself. Many men who are competent practitioners or competent laboratory men are not competent teachers.

I once lived with a man who was a splendid horseman. I never knew a man who could go out into the country and match up a team of horses as he could. He would see a gray colt in this county, and perhaps thirty miles away he would see another one, and he would put them together and they would match beautifully. But he could not teach that art to anyone else. I used to ask him how he did it. He did not know. It was purely intuitive with him. He had not the first instincts of a pedagogue.

Then, finally, you men in the field should use your influence toward helping these state veterinary schools to become more adequately equipped, adequately manned and better provided for. I can remember when I first took hold of the reins at Ohio State, that the members of the State Veterinary Association could not be induced to come onto our campus. I invited them year after year for three or four or five years before they would meet with us. They seemed to feel that we wanted their offices or that we had some ulterior motive. I think that is the reason the cannibal eats the Methodist minister when you send him there. He believes that the minister has some ulterior motive, and so he puts him out of the way, and at the same time he enjoys a good banquet.

We offered them building space and everything of that sort, and finally they came; and see how the influence developed. At the first meeting I attended of the State Veterinary Association of Ohio there were nine men in a bedroom in the hotel, upstairs. Five of them were sitting on the bed, one was sitting on his haunches, and the rest of them on what few chairs there were. And of the nine men three were horseshoers and not veterinarians at all. At the meeting we had there last winter there was an attendance of nearly 500, not all members of the association, however, and they had a splendid time and a good program, and they are right with us.

And that is the spirit which I believe should exist every-

where. I told them, I said, "Gentlemen, the more you boost this institution the more it indirectly will boost you. The greater we become and the more recognition we receive from the people of the state, the more recognition you will receive. What we do is really an index of what you and we are to become."

A great many feel that once they have entered our profession, that suffices, that the profession will make them. But, gentlemen, they surely have the cart before the horse in this regard. No profession can make a man, but men do make professions.

In this rambling chat, for which I have held you gentlemen on this sweltering September night, I hope you may get at least this one thing out of it, which you may retain, that we want to get into our profession the same type of men and get them in greater numbers than they have come into it heretofore, that are now being shunted into other professions.

I thank you most cordially for your attention. (Applause.)

Dr. C. M. Haring was then introduced by Dr. Dimock and spoke as follows:

Dr. Haring: I am afraid Dr. Dimock has made a mistake in commending me to your approval as a competitor with Dr. White. I feel highly honored to be asked to say something to you, but I feel that there are many here who are better qualified in the ability to speak, and in practical experience and knowledge than I am. When Dr. Bemis spoke to me this afternoon about appearing tonight I felt highly honored. I did not feel like refusing, but I have been casting about ever since for something to say, and I had decided to wait until the evening for the inspiration. When Dr. White began telling that story about the negro preachers, one of whom prepared his sermons and the other depended upon inspiration, I felt that perhaps it might have some application to me. But I am afraid that I will not be able to fill the bill as acceptably as did the man who preached the sermon on the "pill."

It reminds me of a story somewhat similar to that, about two negro preachers, one of whom prepared his sermons, and the other one spoke extemporaneously. The first one explained that he always prepared his sermons, but the other one said he used to, but that he did not any more. He had an idea that the devil might look over his shoulder and would know what he was going to say before he said it. So he said, "Now I speak extempore, and the devil himself don't know what I am going to say." (Laughter.) So I feel in about the same position.

I have fortified myself a little with some statistics which

I prepared during the last year, not for the purpose of this address, but as secretary of the A. V. M. A. I thought perhaps they might be interesting to you in connection with what Dr. White has said this evening in giving us an interesting history of the development of veterinary education in America. These statistics show somewhat the condition of the veterinary profession in America at the present time.

At the meeting at Detroit, Dr. Mayo, the chairman of the committee on intelligence and education, made the statement that there were 3,240 veterinary students in the United States at the present time. That certainly is to me a rather astonishingly large number. I am told that that is a larger proportion in comparison with our population in this country than exists in any other country in the world, at least any European country. However, perhaps our needs for veterinary service are and will be greater. There are 835 seniors in the veterinary colleges of America who graduated last June. The number was never so large before.

In view of the inroads of the automobile upon the veterinary business in some sections, the question has been asked, What are these men all going to do? On the Pacific coast I think perhaps we are feeling the effects of the automobile more than throughout the Middle West and the East. I am pleased to learn, almost everywhere I am told that the veterinary practice is good, that there seems to be a demand for more veterinarians.

In compiling some statistics concerning the number of veterinarians, I was interested to compare them with the amount of live stock in the country, and although in California we have a less number of veterinarians than in some of the states, in comparison with the live stock, our live stock industries are so arranged that they in a way do not demand the veterinarian as much, perhaps, as they do here in Iowa. The range cattle business is not one in which the veterinarian can be of as much assistance as in this section where the business of live stock production is carried on in a more intensive way. We have centers of population there where the veterinarian depends upon canine practice, and upon horse practice, and of course the horse practice has been injured somewhat by the inroads of the automobile, and we feel that we are sort of still in the period of development that Dr. White has explained, where the range animal is still predominant, so far as the bovine is concerned.

I stopped off here to learn what I could, to take it back to California. The State Veterinary Association has asked the university to do something for the veterinary profession. We have been teaching the laity, teaching agricultural students

veterinary science to the best of our ability, for about ten years. And there has been some objection to that, perhaps well founded. We are obliged to teach it, however, and we do it as well as we can. But the veterinarian has said, "What are you doing for the veterinary profession?" The university authorities have suggested that we might begin perhaps by giving a short course. That is one of the reasons why I am here, to get pointers on a short course.

Of course, the live stock world looks to Iowa, which is the greatest live stock state, to lead in progress, not only in live stock production, but in live stock conservation, and I believe that the veterinarians of Iowa are living up to the requirements of the state. Certainly I have profited by stopping here.

Now, take Iowa. I had an opportunity to secure the number of veterinarians in every state who are registered under the Harrison Narcotic Act. That presumably includes those who are in active veterinary practice only, and presumably those who are licensed men, although I believe in some states the federal authorities do admit men under the Harrison Narcotic Act who do not hold a license. That depends somewhat upon the state law. In California I believe no one can get a license under the Harrison Act unless he is a licensed practitioner. Perhaps that is so in Iowa.

You have in Iowa 650 practicing veterinarians, licensed under the Iowa Practice Act. You have in Iowa $393,003,000 worth of live stock. That is the greatest value of live stock in any state. This is the census of 1910. That represents $604,620 worth of live stock for every practicing veterinarian in Iowa.

You may be interested to know what proportion that is as compared with other states. Of the forty-eight states I have listed here, Iowa stands eighteenth in that respect. That is, Wyoming has a less number of veterinarians than any state in proportion to the value of her live stock. They have thirty-eight veterinarians in that state, and they have $65,000,000 worth of live stock; that is, as estimated by the census. That is, $1,726,473 worth of live stock to each veterinarian. Obviously the conditions of live stock raising in Wyoming are quite different from those in this state, but there seems to be no reason why there should be a less number of veterinarians there.

There are in the practice in the United States at the present time, registered under the Harrison Narcotic Act, 11,576 veterinarians. This does not include the majority of those who are in the Bureau of Animal Industry. Three thousand,

two hundred and forty veterinary students, and 11,576 practitioners.

I had an opportunity of talking with President Pearson this afternoon, and I seized the opportunity to question him as to whether he knew if any attempt has been made by educators or those who administer educational institutions, to determine the needs of professional education with respect to the number of professional men engaged in the profession; that is, how many students should be graduated in medicine each year in proportion to the number in practice, thinking that I might get some points of interest. He said that so far as he knew —and this was backed up by one of the persons who seemed to be somewhat of an authority on the subject—as far as he knew no attempt had been made to estimate the needs. He felt that it was more or less a matter of supply and demand.

I was very much interested in President Pearson's statistics that he gave last night, of the $300,000,000 worth of live stock lost through diseases annually in this country. That is nearly as much live stock as exists in the entire state of Iowa. I remember he estimated that if the veterinary practitioners would save 50 per cent of this, and that in their fees they would divide with the owner of the live stock fifty-fifty, they might have 15,000 veterinarians at $10,000 a year. I believe he said 15,000 more, but I believe it would figure out about 15,000 veterinarians at $10,000 a year.

These statistics have been published on page 613 of the July number of the Journal of the American Veterinary Medical Association.

I believe it would be to the advantage of the veterinary profession to give some thought as to the number of veterinarians and the quality of the veterinarians in this country, and to organize to protect themselves and to obtain recognition.

If I have any message for you tonight which may be helpful it is along the line of the possibilities of veterinary organization. Dr. White has expressed to you the needs of organization. Of course, I am somewhat opinionated in the matter, having been connected with the A. V. M. A. rather closely during the past year. I have felt that the A. V. M. A. has the possibility of being made an organization of great value, not in a general way, but in a specific way, to every veterinarian in practice in the United States.

During the past year you may be interested to know that Iowa had the largest number of applicants for membership in the A. V. M. A. of any state. There were thirty-five admitted from this state. That number is largely due to two of the faculty of this institution, and to Dr. Hal Simpson,

resident state secretary in this state. Iowa has at the present time, or had, seventy-eight veterinarians who were members of the A. V. M. A., out of the 650 who are resident in this state; and thirty-five being added makes 113 members of the A. V. M. A. in this state. It stands thirty-eighth in the list of states. It seems as if it would be desirable that the greatest live stock state in the Union should be better represented in proportion.

Pardon my reference to California, but I am proud to say that California until year before last stood first in the number of members in the A. V. M. A. It now stands third in proportion and also in the actual number of members. We have in that state 371 practitioners, and we have 140 members in the A. V. M. A. That is, we have actually more members in the A. V. M. A. than any other state, not only proportionately, but actually. At the present time New York stands second with 152, and Pennsylvania stands first with 154. New York has 637 practitioners and Pennsylvania has 548 practitioners registered under the Harrison Narcotic Act, while California only has 371.

Dr. White: What has Ohio?

Dr. Haring: Ohio has 682 practitioners and it has 105 members in the A. V. M. A. That is, the percentage of membership is 15.3, or was before this last meeting when the applicants were admitted. Ohio has had a fairly large number of applicants for membership, but not as many as Iowa.

I believe that it is only through organization that we can obtain the recognition which the veterinary profession needs and deserves in this country. It is only through organization that we can advance the interests of our profession, and incidentally our own personal interests. I believe that the state association is the place to form the unit, that that should come first. I believe the A. V. M. A. should co-operate with the state associations. I hope eventually the state associations and the A. V. M. A. can be amalgamated into one organization, and steps have already been taken in that direction.

When Dr. Archibald was elected president, one of his first acts was to appoint as resident state secretaries in the various states, the secretary of the local state association. This seemed to be in most states very acceptable, and I believe has been one of the reasons why the number of applicants for membership in the A. V. M. A. this past year has exceeded by nearly two-fold that of any previous year. The purchase of the Journal has added greatly to the prestige of the association. This growth has been in spite of the fact that the dues of the A. V. M. A. have been raised to $5 from $3. Al-

though you can get the Journal for $3, you have to pay $5 annually to be a member of the A. V. M. A., thus putting a premium upon membership. That is, a man who is not a member can get all the proceedings for $3. I was very much disappointed that the association did not continue the dues at $3, because the association is in good financial shape. The Journal has been purchased outright, with the exception of a $500 note which remains to be paid, and does not mature until November, and there is plenty of money in the treasury to pay that. Of course, the money which will accrue from the $5 memberships can be used to great advantage, and I trust that it will be. Plans are already being laid to use the surplus in tests of veterinary remedies, in securing uniformity in legislation, and in organizations to direct public opinion for the benefit of the veterinary profession, and if it is handled properly this surplus no doubt can be used to great advantage.

I was not disappointed because we are going to have this extra money, but I felt perhaps if the dues were not raised, the association might develop into a large association. We ought to have at least 7,000 out of the 11,000 practicing veterinarians in this country as members of the American Veterinary Medical Association. I think that it probably will grow to that in a few years, but what a force for help such a strong organization could be. Nobody gets out of the A. V. M. A. what he does not put into it. You have got to give in order to receive. But I believe that through state organization and national organization the veterinary profession is going to be advanced more rapidly than in any other way.

I thank you. (Applause.)

SHOEING AND BALANCING

DR. J. W. ADAMS, UNIVERSITY OF PENNSYLVANIA

According to the topic assigned me on the program, I am supposed to speak on "Shoeing and Balancing." If I speak about shoeing in its completeness, of course it will require thirty or forty hours to go over the subject even hastily. That I cannot be expected to do in this short time at my disposal. I hope to enunciate certain principles that may be applied to shoeing and which are applicable to each individual horse.

To understand properly shoeing, we must first have some knowledge of the shape and structure and growth of the hoof, and I will devote myself for a few moments to a consideration of this question.

As you know, the hoof is an epidermal structure, like the cuticle of the skin or the hair of the head. It is insensitive, in that it contains no sensitive nerves. It contains no sweat glands nor blood vessels. It is simply an insensitive, dry, tough protection to the lower part of the foot. And yet it has certain movements transmitted to it by the vital structures within, and it has a certain definite shape in each individual, depending largely upon the direction of the leg above. There ought to be as many differences in the shape of hoofs as there are in the features of the human being. Among ten thousand faces you never find two that are alike, and probably among ten thousand hoofs you will never find two that are exactly alike.

The shoe which is designed to protect the hoof against inordinate wear, or to provide the hoof with a means for gripping the ground and preventing slipping, should be shaped in accordance with the normal shape of the hoof. Therefore, it is necessary first for the horseshoer to have a very distinct idea of what the shape of each hoof that presents itself to him should be. If there are normal shapes of feet, which depend upon the slant of the limbs above, there should be many forms of shoes designed for those different shapes of hoofs. It is wholly irrational for a man to use shoes that are symmetrical and apply them to all the different styles of feet without shaping each branch to conform to the natural curvature of the walls to which it is to be applied.

First, we will discuss the shape of feet, depending on the normal standing position of the horse; let us consider the standing position of the normal horse viewed from in front, because we may view these legs from in front or in profile or from behind. This chart which I present to you was drawn

from life. The chart represents an anterior view of a draft horse, and you will note that the legs are perpendicular from the shoulder joints to the ground. That is, if a perpendicular be dropped from the point of the shoulder to the ground it will apparently cut the foreleg into an inner and outer half of equal width, going through the center of all the joints, and meeting the ground in the middle of the toe. That is said to be regular or normal. That is the normal standing position of all horses, whether they be trotting-bred or draft-bred, as viewed from in front. The legs from the shoulders down should be perpendicular, and the toes should point straight ahead, neither to the outside nor to the inside. The feet, the hoofs on the ground are usually wide enough apart so that a hoof of the same size may be placed between the two; with these heavy draft horses, wide in the shoulders, the legs are straight at the top and wide apart, but the foot also is a large, round, wide foot and fills in the space between the hoofs, and would just about occupy that space between the hoofs.

Now, there are deviations of the whole leg to the outside, or part of the leg to the outside of the perpendicular. There are deviations of the whole leg to the inside of the perpendicular, or deviations of a part of the leg to the inside of the perpendicular.

In this chart (referring to same) is shown the base-narrow standing position, in which position the leg from the shoulder joint downward deviates to the inside of the perpendicular. In this case, of course, the perpendicular is not shown, but if you draw a perpendicular line from the point of the shoulder to the ground it naturally would fall to the outside of the leg. In other words, the direction of the entire leg from the shoulder joint to the ground is downward and inward. The feet are placed too close together, and the toes may point straight ahead or may point slightly inward. This is the base-narrow standing position.

In this chart (referring to another chart) the legs from the shoulder down deviate to the outside of the perpendicular. The feet are placed too wide apart on the ground. The toes usually point slightly outward. This is the base-wide standing position.

I show you these three standing positions because the shape of the hoofs in each of the standing positions is different and should be different and should be kept different from each other, and should be shod differently.

This (indicating another chart) is the toe-narrow standing position. The legs from the shoulders to the fetlock are perpendicular, but below the fetlock the phalanges pass downward and to the inside of the perpendicular, bringing the

hoofs too close together, and directing the toes inward, so that this animal stands pigeon-toed, or toe-narrow. And the shape of the foot is much as in the base-narrow standing position. You will notice that in the pure base-narrow standing position that the phalanges run down and in, in the toe-narrow position, they run downward and inward, but more pronouncedly than in the simple base-narrow standing position. That is a deviation of a part of the leg to the inside of the perpendicular.

In this chart (indicating) the animal stands toe-wide. The legs from the shoulders to the fetlocks, are perpendicular, just as in the normal, but below the fetlock the deviation of the phalanges is downward and outward, and we call that the toe-wide or splay-footed standing position. And the shape of the foot, because the phalanges run downward and outward, is very similar to the hoof of the base-wide standing position.

In this case (indicating another chart) the knees are too close together, the fetlock joints are too close together. The phalanges run downward and outward, and the animal is said to be knock-kneed, or close between the knees, and toe-wide. That is a modification of the base-wide standing position.

In this chart (indicating another chart) the knees are too far apart. The phalanges run downward and outward. The shape of the foot is that of the toe or base-wide standing position. This animal viewed from the side will be bound to be knee-sprung.

This (indicating) is a chart showing the knee-narrow or knock-kneed standing position, and the toe-wide standing position.

Now, let us view the hind legs from behind (referring to a chart). A horse stands normally, when viewed from behind, when a perpendicular line dropped from the tuberosity of the ischium to the ground cuts the hind legs into inner and outer halves of equal width, cuts through the center of the point of the hock, to the center cannon of the fetlock, and meets the ground opposite the middle cleft of the frog. In other words, the hind leg viewed from behind is perpendicular all the way down. That is the normal standing position behind; and we have deviations to the outside or to the inside of that perpendicular as in the front legs.

In this chart the legs pass downward and outward and we have the base-wide standing position.

Here the hind legs (referring to another chart) pass downward and inward. The feet are too close together, and the base-narrow standing position is represented.

This chart (indicating another chart) represents the bow-legged standing position. The hocks are too wide apart. The

feet are too close together, and the toes are turned inward. The animal is wide between the hocks or bandy or bow-legged, and base or toe-narrow below. This is quite a common formation in heavy draft horses, wide in the hips and widening out behind. When such horses walk along they pivot on the toe, and the heel swings out. They scour their toes out rapidly by that twisting movement.

(Referring to another chart.) Here is an animal close-hocked, the fetlocks close together, the feet close together, and which is toeing straight ahead and slightly inward. That is the close-hocked standing position.

(Referring to another chart.) Here is a close-hocked horse, which is toe-wide below.

(Referring to another chart.) Here is the so-called cow-hocked horse, where the whole hind leg, from the stifle to the ground is rotated outward. The hocks are not only close together, but the points of the two hocks are directed toward each other. The fetlocks are wide apart, the phalanges run downward and outward, and the animal stands base-wide and toe-wide.

The shape of the foot is determined also by the slant of the pasterns, viewed in profile. We recognize three important slants of the pasterns viewed from the side. The foot axis is an imaginary line passing through the long axis of the three phalanges. In this chart (illustrating) the foot axis is shown by a dotted line drawn from the surface of the foot; the dotted line begins in the center of the fetlock joint and is carried in a general direction to the long and short pastern, to the coronary band, and thence prolonged to the ground, parallel to the line of the toe. In this case the foot axis is straight, and the three phalanges are end to end, forming a straight column. That is normal.

The chart also shows the relation of the slant of the pasterns to the ground surface. Some feet stand very steep in the pasterns, other stand quite slanting in the pasterns, and it is impossible to estimate what slant is considered as normal. The slant of the fore pasterns differs from the slant of the hind pasterns. The slants of the fore pasterns in profile are normal when they make an angle with the ground of 45 to 50 degrees, when the angle between the foot axis and the ground is from 45 to 50 degrees. You know that a right angle is an angle of 90 degrees. Half of a right angle would be 45 degrees. A horse standing normally, as viewed from the side, when the fore pasterns make an angle with the ground of from 45 to 50 degrees. The slant of the hind pasterns is slightly greater, being normally from 50 to 55 degrees.

Here (indicating) is shown the front pasterns in profile, in which the pasterns are relatively upright, and the angle between the foot axis and the ground is greater than 55 degrees. That is the upright or stumpy pastern, and the hoof that is joined to it is an upright or stumpy hoof.

In this chart (indicating) the angle between the pastern and the ground is less than 45 degrees. The pastern is quite slanting. The fetlock is low, and sinks still lower under the weight; and the hoof that is joined to it is the sharp-toed hoof known as the acute-angled hoof.

Now, let us consider the shape of the foot as dependent upon the slant of the foot axis. In the foot axis of normal slant, from 45 to 50 degrees, the perpendicular lies through the leg, and just about touches the bulb of the heel. The toe line should be straight, neither bent in or out, and the heel line should be parallel to it. As you look at the bottom of the foot (referring to another view). This represents the bottom of the leg displayed above—we will notice that the outer half of the sole is a little wider than the inner half of the sole, that the outer quarter is more sharply bent than the inner quarter, that the wall around the toe is thicker than the wall at the quarter, that the branches of the frog seem equally developed, and that the heels slightly overhang the buttresses of the foot.

In the acute-angled foot the outer half of the sole is wider than the inner, the outer quarter is slightly more curved than the inner, the branches of the frog are equally developed, and the bulbs of the plantar cushion considerably overhang or project back of the buttress. Compare these two feet and see how much farther back the buttresses project in the acute-angled foot than in the normal foot. That has some significance, because where the bulb of the plantar cushion projects farther back in the foot, the branches of the shoe should project farther back than the buttress to support that overhanging heel.

Here (indicating another chart) is shown the upright foot, where the angle between the foot axis and the ground is greater than 55 degrees. Note how square and short the foot or solar surface of such a hoof is. The outer branch of the sole is still a little wider than the inner, but not markedly so. The buttresses are wide apart, the bulbs of the plantar cushion project but slightly back of the buttresses.

Now, a word or two as to the flight of the foot viewed from in front and from the side. In the normal standing position, as the horse moves toward the observer, the feet should apparently rise and fall in a vertical line. When the horse is in a normal standing position, that is, when the legs are perpendicular and the toes straight ahead, as he walks or trots toward

the observer, each foot should simply rise and fall in a vertical line, that is, should be carried neither out from nor in toward the median line.

In the base-wide standing position, the foot leaves the ground at the inner toe, is then carried forward, and inward, to support the leg, and then outward and forward to the ground, which it meets on the outer toe. Therefore, the foot executed a circle, and to the observer placed in front, rises and swings in toward the supporting leg, and then drops downward and outward to the ground.

In the base-narrow standing position, the foot breaks over either center of the toe, swings forward and outward, out to support the leg, and then forward and inward, meeting the ground with the outer toe. The foot, therefore, executes an outward swing, and the animal is said to paddle.

The flight of the hind feet, viewed from behind, is the same as the flight of the front feet viewed from in front. That is, in the normal standing position the feet rise and fall in a vertical line. In the base-wide position they pass forward and inward, and then forward and outward; and in the base-narrow position they swing outward in a circle.

The flight of a front foot, viewed from one side is dependent chiefly upon the breed and the nervous activity of the horse, but to a slight extent is modified by the weight of the shoe and the slant of the foot axis, viewed from the side. With the normal slant of foot axis, the front foot leaves the ground at the toe and rises gradually until it reaches its highest point when it is opposite the supporting leg, from which point it gradually descends to the ground. Some horses are bred to be high steppers, and will pass the supporting leg at a higher point than others. You know some horses travel low and some naturally travel high. And the height of the action is determined partly by the breeding and partly by the dressing of the foot and partly by the weight of the shoe. The long slanting toe, which makes it difficult for the horse to break over, starts the foot upward, and makes the animal travel higher than if the foot has the normal length of toe. So that the long toe tends to a certain extent to increase the height of the stride. On the other hand, the short toe, which makes it easy for the animal to break over, starts the foot in a direction more or less parallel with the ground and favors low action. Now, increasing the weight of the shoe in the long-toed horse, adds still more to the upward fold of the pastern, and increases the height of the action; whereas, the light shoe, on the upright or stumpy foot, adds very little to the height of the low action of this foot.

The effect of weight upon action is always to increase the

centrifugal force of the foot. Let us explain what we mean by that. Take a string a yard long and put an ounce weight on the end of it and swing it around your head. There is a pull from the center which we call centrifugal force. If we put a two-ounce weight on that yard of string and swing it around at the same speed, it pulls with twice the force outward and tends to fly off on a tangent. Suppose, then, we are dealing with a toe-wide horse, one which swings its foot inward close to the supporting leg, and then outward to the ground, and shoe that foot with a heavy shoe. The heavy shoe increases the centrifugal force of that foot, moving in a circle, and tends to make the foot move in a wider circle. So wide, possibly, that it brings it in contact with the fetlock or the knee.

Or, suppose the foot moves outward in a circle, that the animal paddles. The heavy shoe increases the centrifugal force of that foot; the tendency of that foot is to fly off at a tangent, and makes the animal's foot execute a still wider circle and makes him paddle worse. It makes very little difference where the weight is placed in that shoe.

Let me talk a little faster and give you an example. I suppose you are familiar with it, but these are good principles which we want to develop first. A friend of mine was called on to shoe a big, wide-breasted draft horse, that was pigeon-toed and who rolled. He swung his feet out so far. He thought: "I will just try to make him travel in a line, and I will put the weight on the inside branch of the shoe." He got him shod with heavy shoes, with the inner branches weighted, and he just rolled worse. He said, "That is funny. I thought that that weight on the inside of the foot would straighten him out." "No," I said, "weight anywhere in that foot is bound to make him swing around a wider circle. Weight on the inner branch will not do as much harm as weight on the outer branch. And why? When the paddling foot moves around in a circle the center of the circle is over on the other side. It may move in a circle around a radius of, we will say, four feet. If a weight is placed on the outer branch of the shoe, in that case the radius of the circle is four feet plus the width of the foot, which is four inches. That is a longer radius, and therefore, weight on the end of the longer radius is moving at a greater speed, and therefore there is greater centrifugal force. So that in a foot moving in a circle, if the weight is on the outside of the foot, there is greater centrifugal force than if the same weight is on the inner branch of the shoe, because on the inner branch it is attached to a shorter radius, which makes a smaller circle.

Remember, then, that when the foot is moving in a circle

outward from the median line or inward, increasing the weight increases the tendency of the foot to fly out at a tangent; in other words, to increase the centrifugal force. The principle is that when the foot moves in a circle and we wish to straighten the circle or make it less pronounced, the weight should be cut down to the minimum.

Now, suppose we wish to increase the height of the stride by adding weight, and weight does do that. Viewing the front foot from the side, it rises first, and if the weight of the foot is increased by a heavy shoe, when the foot is started with a certain speed upward, the momentum due to the increased weight carries the foot a little higher before it tends to drop. Of course, the added weight is tiresome to the flexor of the forearm, and such an animal rapidly tires, but while strong and fresh, the weight in the foot does by increasing the centrifugal force, carry the foot a little higher than it would otherwise be carried. And the weight in such a case should be placed as near as possible to the toe, in order to increase the centrifugal force as much as possible.

Let us next consider the shape of a normal front foot in the regular standing position, viewed from in front (referring to a specimen). This is the near front foot. Viewed from in front, the outer wall should be slightly more slanting than the inner, but should be straight from the coronet to the ground, neither concave nor convex. Viewed from below, the outer half of the sole, from the point of the frog should be slightly wider than the inner half of the sole. The outer quarter should be slightly more curved or bent than the inner quarter. Both branches of the frog should be equally beveled.

In the base-wide foot (referring to a specimen) the outer quarter should be considerably more slanting than the inner quarter. The outer half of the sole, considerably wider than the inner half of the sole, the outer quarter distinctly more curved than the inner quarter. In the base-narrow standing position the inner quarter should either have the same slant or a greater slant than the outer quarter. In other words, the outer quarter stands steeper than normal, and the inner quarter stands more slanting than normal. When viewed from below, the inner half of the sole is wider than the outer, and the inner quarter more curved than the outer. The outer quarter is relatively straight when compared with the inner quarter. The inner branch of the frog is better developed than the outer.

Next, a few words as to the physiological movements of the hoof or all those movements which take place within the hoof when the weight is cast upon it and taken off. The hoof is not a dead, insensitive and immobile structure, but it is

highly movable in health, and scarcely any part of the hoof does not undergo some slight change of position when the weight is put on or taken off the foot. These slight changes of position we term the physiological or normal or healthy movements of the hoofs. When the weight is thrown on the leg, is transmitted downward and forward, through the three phalanges, it causes first a descent of the entire os pedis or coffin bone. This descent carries the sole downward and flattens it slightly so that it is less arched than it is when not bearing weight. It flattens the sole and draws the wide walls in at the coronary bands, so that the foot becomes narrower from side to side through the coronet, widening the sole at the plantar or ground surface. Then, as the short pastern rotates backward, it presses the tendons down into the plantar cushion, and the plantar cushion, resting upon the ground, is squeezed from above to below, and like any elastic body, tends to dilate laterally. The expanding plantar cushion lying in contact with the lateral cartilages presses them apart and carries the walls of the quarters farther apart and expands the whole posterior third of the hoof. The fleshy frog and the horny frog are also compressed and flattened from above below, and widened from side to side. These are a few of the physiological changes that occur within the hoof.

Now, what are they? The descent of the os pedis, the flattening of the sole, the flattening of the frog, the narrowing of the foot from side to side at the coronary band, and the widening of the bottom of the foot from side to side at the ground or shoe surface, and the expansion or widening of the whole posterior third of the foot, most marked, of course, at the plantar border.

Now, this widening of the posterior third of the foot is best noticed, of course, upon examining the shoe, a plain shoe, which has been worn for three or four weeks. In removing such a shoe we should find brightly polished grooves upon the upper surface of the shoe. These are due to the scouring of the horn against the shoe, to expansion under weight and contraction when the weight is taken off, assisted, of course, by grit and sand working in between the hoof and the shoe. So that all plain shoes, calked shoes and bar shoes, after a certain amount of wear should show these polished grooves, because they indicate expansion and contraction of the normal healthy quarter.

Now, the dressing of the hoof for the shoe is an important part of shoeing, and in my opinion it is the part of the process which receives the least attention from the horseshoer, the dressing of the foot. The dressing of the foot means the preparation of the hoof to wear the shoe. It consists in short-

ening the walls so that the foot shall be placed to the ground flat, and the inner and outer walls shall touch at the same time. If the horses, for instance, were allowed to travel bare-footed over perfectly level cement or sandstone, they would dress their hoofs perfectly. The long parts would hit the ground first, and the greater scouring they would receive. The high parts would be cut down until finally the whole foot at a walk or a trot would be set down flat on the ground, just as nature intended that it should be. But the shoe, the plain shoe prevents this rapid and proper leveling of the walls.

In dressing a hoof the first thing to do always is to remove the dead sole. The loose flakes are removed, and then the horn of the sole may be pared, but not so far as to remove all of the little lines and creases, which Nature forms, to undermine and exfoliate the useless horn. The foot which I hold before me (referring to a specimen) shows one-half properly dressed and the other half overdressed. If we open the outside of this foot, the outer branch of the sole, all the dead flakes that are to come away have been removed, but we can still see little cracks which show that Nature is beginning to work down and exfoliate the horn. The sole normally should not be dressed until these small cracks have entirely disappeared. Sometimes it is necesary for surgical purposes in diseased conditions of the hoof to dress until we get down to soft yielding horn, but when such moist horn is exposed, by the horseshoer's rasp, evaporation takes place and the horn rapidly dries and contracts, and damage is done to the pododerm underneath.

The frog should have its loose rags and tags removed, but should always be left prominent enough so that it projects above the surface of the wall, and if a plain shoe is to be applied, the frog ought to project an amount equal to the thickness of the shoe, so that when the foot is placed on the ground the frog ought to touch, even though the shoe is in place. Of course, the luxuriance of the frog depends upon the breeding of the animal. We have trotting-bred horses, for instance, which have very small frogs, that grow very slowly, and in such an animal the frog often hardly produces horn enough to reach the ground when a plain shoe is on, but the object of the frog is to strike the ground as a soft cushion and take the shock from the joint surfaces and tendons, and therefore, it should be less prominent and kept soft. So the frog should not be overpared. The loose flakes should be removed, but it should be left prominent.

If we are to put a bar shoe or a rubber pad upon the foot, the frog must not be left too prominent, or the animal will get too much frog pressure, and an inflammation of the

plantar cushion will result. For instance, in dressing a foot for a bar shoe, the frog should be left just so prominent that when the shoe is laid upon the foot it can be pressed by thumb pressure far enough to bring it in contact with the wall of the quarter.

How prominent shall we leave the frog if we are going to put on a bar shoe or a rubber pad? Only so prominent that when the loose shoe or the rubber pad is placed on the foot, by thumb pressure we can spring the shoe clear down to the buttress and make it touch. If there is so much horn that you cannot do it with a bar shoe, you are giving too much frog pressure, and he is going to get sore in the plantar cushion.

I remember one day in the Military Shoeing School in Dresden I saw the shoes removed from a pair of Belgian horses, about five or ten minutes before twelve, and then the men went to lunch, and I came back just before one o'clock, just before the man began to work, and one of those horses was lying on the floor, and the other fellow had shifted his weight, standing on his hind leg, and tried to get his weight from one leg to the other. This horse had been shod with toes and heels and had prominent frogs, and the frogs were so prominent that the shoe could not touch anything but frog. One horse had gotten so sore in an hour that he lay down in order to get the weight off the frogs, and the other horse was balancing back and shifting his weight from one foot to the other. That is an example of what extreme frog pressure will do.

The shortening of the wall follows the dressing of the sole and frog, and that portion of the wall which projects beyond the surface of the dressed sole may be removed with the hoof rasp or the knife. In country shops where the feet are long they use an instrument known as the butteris, which is simply a sort of chisel. The butteris is all right, if used moderately, but it makes the cutting so easy if the foot is soft, that the operator is very apt to overcut. The rasp and the hoof knife are instruments used for shortening the wall. The wall is shortened until it is brought on a level with the previously dressed sole.

In shortening the wall we should always have in mind the wear of the old shoe. By its wear or in its wear is a good record and a true record of the manner in which that foot has been set to the ground during the period when that shoe was worn. When a horse is to be shod we pull off the old shoes, and instead of throwing them to one side it is better to give the shoe a careful examination before it is thrown away and if we find that the inner and outer branches are worn down about equal distances, that is an evidence that the horse has been

putting his foot flat with the ground, while he was wearing that shoe, and that he got just about the same amount of scour on one branch as on the other. It is evidence that he was properly balanced, that the relation in length between the inner and outer walls was correct, and we wish to shorten the foot so that that relation will be maintained and the inner and the outer walls will strike at the same time.

Now, suppose on the other hand, the old shoe showed increased wear on the outside, that the outer branch was much thinner than the inner, what is that evidence of? It is an evidence of the fact that the wall above the thin branch was too long, that the shoe was tilted down on the long side, and reached the ground first, and got a considerable scouring before the inner branch touched, so that shoes showing uneven wear are evidences of too great length of wall above the more worn part of the shoe. So that it is well always to dress the foot in accordance with the wear of the old shoe.

The wall is shortened and leveled. Half of the wall should be shortened, and then with the rasp it is leveled until the wall is dressed horizontal from the outer edge in to about one-eighth of an inch or three-sixteenths of an inch inside of the white line, so that only the thickness of the wall and the white line and the narrow margin of the sole shall rest upon the upper horizontal surface of the shoe, which we call the bearing surface of the shoe. The wall and the white line and the narrow margin of the sole should rest upon the shoe. If the sole is cut down so that it does not touch the shoe and the animal is heavy, the feet are wide and the sole rather flat, there is always a tendency for the sole to descend if it does not touch the shoe, and for a crack to form in the white line, and we have then what is known as separated wall, a crack which reaches the lower ends of the fleshy leaves and makes the animal sore. The sole should rest by its margin a quarter of an inch in a big foot, and three-sixteenths of an inch or one-eighth of an inch in a small foot, upon the shoe. The resting of the margin of the sole on the shoe does not cause any bruising of the pododerm and does not give rise to corns or bruised sole, but is normal and natural.

The upper or thin edge of the wall may be broken away with a rasp. But this corner should not be too deeply rasped here (indicating). Otherwise the white line is brought too near the outer margin of the sole, and we have too shallow a hold for the nails.

How much shall we take away? We take away one-half of the margin of the wall to make the absolute thickness of the wall equal to the relative thickness. You probably will not understand what I mean by that. I will have to explain it by

a drawing (referring to a blackboard drawing). We will take an acute angled foot and consider these (indicating) the fleshy leaves, and this (indicating) the white line. Now, what is the absolute thickness of that wall? The absolute thickness is the distance from the surface to the fleshy leaves, measured at right angles or perpendicular to the surface. We might say the absolute thickness in this case is one-half inch. What is the relative thickness? That is, the thickness of the wall measured on the sole? And that distance would be more than half an inch. It might be five-eighths of an inch. That might be five-eighths, while this is four-eighths. So the relative thickness of the wall is one-eighth of an inch greater than the absolute thickness. The amount we take off at that corner is the difference between the absolute and the relative thickness of the wall. So that when the toe has a decided slant in an acute angled foot, we always take away more than if the foot is upright. If it is normal, we would take off a little, and if it is acute angled we would take off more. We still leave enough on the bottom for the bar shoe which equals the absolute thickness of that wall, and if we go beyond that we leave too little. Here on this side (referring to the specimen) the edge has been broken away until the relative thickness equals the absolute thickness. On this half of the specimen (indicating) the rasping has been still greater and the white line where the nails penetrate is too near the margin, and the bearing surface of the wall is diminished by overrasping or cutting away the wall of the quarter.

I might say a word or two about nailing. The horseshoe nails are used, of course, to hold the shoe to the foot. Their number depends upon the use of the animal, and their distribution should be such that the nail interferes as little as possible with the physiological movement of the foot. The number of nails runs from eight to five; five nails in a very light, plain shoe and eight nails in a heavy shoe, with heels and toes. Plain shoes require less nails than shoes with calks. And shoes with clips require less nails than shoes without clips, because every clip is worth a nail or two. That is, a toe clip is worth a nail or two. A side clip is worth a nail or two. So the use of clips enables us to do with less nails.

Q. Does the toe clip interfere with the downward and forward growth of the horn?

Dr. Adams: The horny growth takes place at the coronary band. Anything which stimulates the coronary band, increases the blood supply to the coronary band, increases the rapidity of the growth of the horn, doesn't it? Anything that you use on your scalp, which increases the blood supply to your scalp, increases the rapidity of the growth of the hair on

your head. Among those things are massage—and massage in a horse's foot means work. Therefore, the horse that works grows the hoof faster than the horse that stands idle. You might use a liniment on your head, which in connection with massage opens up the blood vessels and increases the blood supply to your scalp. On the horse we may use liniments or blisters around the horn and thus increase the rapidity of the growth of the hoof.

But what we do to the surface of the horn has no influence at all upon the blood supply to the structure from which the horn grows. The barber can sear or singe the hairs on your head, but that will not increase the rapidity of the growth of the hair. We can rasp and we can scrape the surface of the horn. That has no influence at all upon the blood supply at the place where the horn is produced. Whatever we do to the dead horn at the toe, by way of nailing or clipping, has no influence whatever upon the blood supply from which that horn grows.

Now, if you ask me whether the use of clips interferes with the physiological movements of the foot, then I would say this: that the foot has no movement at the toe at all, and the toe clip in no manner interferes with the physiological movements of the foot, but the side clip may if placed too far back interfere with the lateral expansion of the quarters.

Q. Does not the clip at the toe of a shoe interfere with the growth of the horn at that point?

Dr. Adams: The clip on the shoe makes it necessary for the shoe to be carried forward with the growth of the horn, so that the horn cannot overlap the shoe. The horn growing down carries the shoe with it. If there is no clip, there is a tendency for the nails to get loose and for the horn to creep out beyond the border of the shoe, whereas if there is a clip, this creeping out cannot occur and the shoe cannot be shifted back. In other words, without a toe clip, when the nails become loose, the shoe is shifted backward. How? When the foot is moving forward with the shoe, and the shoe strikes the ground, there is friction between the shoe and the ground, and the tendency of the hoof is to glide forward over the shoe, and that loosens the nails; whereas, if there is a clip butting against the toe, when the foot, passed forward and downward to the ground, and friction takes place between the shoe and the ground, the hoof cannot slide forward over the shoe, but pushes the shoe with it. The clip takes the strain off the nails and the nails remain tight longer with the clip than without. All the strain is around the nails when there is no clip. There is nothing but the nail to prevent the foot when the shoe strikes the ground, from sliding forward clear off the shoe.

But with a clip there, the hoof presses against the clip and carries the shoe along, and the strain is largely taken off the nails. So that every shoe should have a clip except the lightest running shoes, because the clip insures longer stability to the shoe and requires less nails than a shoe without a clip.

For heavy draft horses that are pigeon-toed or base-narrow, and that strike the outer toe, the tendency of the foot is to glide forward across the upper toe. In other words, for the shoe to be displaced backward and inward, and the outer side clip tends to prevent that. The toe clip tends to prevent the shoe from sliding back, and the outer side clip tends to prevent the foot from sliding forward, and it requires less nails. It is a good thing to get along with the least number of nails possible. Every nail makes a hole and weakens the wall. A clip is as good as a nail or two.

When you find a foot where the horn has grown beyond the shoe you may regard that as a sign of growth above, but it is not. It is only a sign that the shoe has shifted back under the foot, that the foot has tended to slide over the shoe. And where you find the quarters cut over the shoe, that is an evidence that the quarters have extended and gotten wider than the shoe and crept over, but the toe clips do not interfere with the growth of the horn. There is such a thing as setting the toe clip too deeply, and also such a thing as hammering that hot clip back into that groove too strongly. The receptacle in the toe for the clip should never be cut too deeply, otherwise the shoe when fitted is too far back on the foot, and the inside and outside toe nails come clear across the white line and drive into the margin of the sole, and the last nail behind is too shallow. It goes back of the white line and falls into the substance of the wall, but the toe clip itself does no harm.

I have had blacksmiths tell me that quite a proportion of the horseshoers thought for many years that that little notch that we see in the point of the os pedis was the result of the drawing of the toe clip, but, of course, horses that have never had toe clips show the same little notch. (Referring to a specimen.) This is the hind hoof and does not show the notch like the front foot does, because the os pedis, the front of it has a little notch. It is not absorption of bone, from hammering up the toe clip. This (indicating) is the right hind hoof. We know it is a hind because of this point (indicating).

Q. In slight tendonitis do you recommend raising the heel by a heel calk or raising the toe by a toe calk?

Dr. Adams: How shall we dress and shoe the foot in a case of slight tendonitis? In other words, you are speaking of inflammation of the flexor pedis perforans, what we call the

perforans tendon, the one that lies between the suspensory ligament ahead and the superficial flexor behind. What is the influence of dressing the foot upon the tension of the perforans tendon, if the main tension of the perforans tendon be altered by dressing the foot, by lengthening or shortening the toe or lowering or raising the heel? In tendonitis we should aim to remove the strain from the inflamed tendon. If the horse has an inflamed tendon, which has gotten sore during treatment, we ought to remove the tension and the strain. Is that not what we should do?

The member: Certainly.

Dr. Adams: Very well. How shall we tilt the foot, or can we so tilt the foot as to slacken up the perforans tendon? The perforans tendon is attached to the under surface of the os pedis, and passes backward and upward beneath the navicular bone, along the short pastern, to the fetlock joint, and winds around the fetlock joint and passes upward. It is a sling or a hammock. Here (indicating) is the toe on the ground. If I raise that heel and leave the toe on the ground, I carry the navicular bone away from the tendon, and therefore slacken up the tendon. Or, on the contrary, if I take off the heel calk and raise the toe, and carry the toe up in this way (indicating) and bend the toe in that joint, do I not carry the attachment of the tendon up that way, too? Does not that tighten the tendon around the navicular bone? Raising the toe or cutting down the heel draws the attachment of the perforans tendon forward, making it wrap tighter around the navicular bone and the fetlock joint, thus increasing the tension on the tendon. Raising the toe by a shoe which is thicker at the toe and thin at the branches, or shoeing with a toe calk and not with a heel calk, or lowering the heel by cutting away the heel, carries the attachment of the perforans tendon forward and upward and makes it take a short, tight wrap around the navicular bone and increases the tension in the tendon all the way up.

On the contrary, the lowering of the toe by rasping the toe away, or applying a shoe which is thin at the toe and thick at the branches, or putting on heel calks, carries the navicular bone forward and upward so that it does not press so strongly against the tendon, and slackens up the tendon.

So, raising the toe and lowering the heel increases the tension of the tendon. Lowering the toe and raising the heel diminishes the tension of the tendon.

So that when the horse is sore in the tendon, take off the shoe and rasp the toe down and put on a rubber pad thick at the heel, or put on a shoe with heel calks, or if shod with toe and heel calks, take off the shoe and heat up and knock off the

toe calks. That, of course, slackens up the tendon.

Suppose you have a running horse. Where does he get sore? Maybe you have not had much experience with running horses, but the running horse gets sore in the inner branch of the suspensory ligament. That bears a little more weight than the outer; and if you take a photograph of a running horse at speed, with a jockey on his back, with just one leg on the ground, that fetlock is sometimes horizontal or below the horizontal, showing that there is an enormous strain on the suspensory ligament, and they get sore in the inner branch of that ligament. Suppose I am treating the inner branch of the suspensory ligament in a running horse, and I want to slacken it up for a while while it is sore. How can I slacken up the suspensory ligament? I will make the other two tendons tighter, and they will force the fetlock forward and slacken up the suspensory ligament. How do I make the other two tendons tighter? If we slacken the perforans up by shortening the toe, raising the toe will tighten it, and that is what we do. We put on a shoe that is thick at the toe and thin at the branches, or have a plain shoe with a toe calk that raises up the foot. When the toe is pulled up it tightens up the perforans tendon, and the tendon runs in a groove back of the fetlock joint, and when the tendon is tightened up below, the fetlock is pulled forward and the tendon tightens, and he stands steeper in the fetlock, and it slackens up the suspensory ligament. Of course that is only temporary, while the ligament is sore.

The superficial flexor is seldom inflamed from strain of its own muscle. The superficial flexor is a very weak muscle at its end, a muscle whose contractile strength is not sufficient to overstrain the tendon, and the superficial flexor is seldom inflamed except the horse strikes himself and bruises the tendon against something, or directly damages it by hitting it with the toe of the hind foot, for instance. Slacken off the perforans tendon by lowering the toe and raising the heel, slacken off the suspensory ligament by raising the toe and lowering the heel. That is a good point for a veterinarian as well as for a horseshoer. I am glad that it was brought out.

Q. Where should the weight be placed in the shoe to increase the length of the stride?

Dr. Adams: How does weight increase the length of a stride in a horse? Suppose we put weight on the front foot, how does it increase the length of the stride of the front foot? Here, for instance, is a horse walking along without any shoes on at all. Suppose this is the beginning of the phase of flight of the foot. The phase of flight of a horse's foot is the distance that it travels from the time that it leaves the ground

until it touches the ground again. That is the flight of the foot. We divide that space of flight into halves. The path that the foot travels in the air from the time it leaves the ground until it is just opposite the supporting leg is the first half of the phase of flight, and the path which it travels from that point until it touches the ground again is the second half of the phase of flight, and it is necessary to distinguish this flight by halves, because we often find that one-half of the phase of flight is increased or lengthened, and the second half of the phase of flight is shortened. Take, for instance, a horse lame in the shoulder. The forearm is brought forward by the action of the biceps muscle, which is painful, and the horse will hesitate to do it, and he will allow the lame leg to be away back under his body before he will use the sore muscle to raise the forearm. That increases the first half of the phase of flight. The animal hesitates to use his biceps until his body has moved forward, and the foot stays far back under the body, and the distance it has to travel from that point until it reaches the perpendicular or supporting leg is longer than it should be, and we say that the first half of the phase of flight with a shoulder-lame horse is increased. But he fails to step out far. He sets his foot to the ground and the second half of the phase of flight is much shorter than it ought to be.

Now we are ready to develop the next point. An increase of weight in the shoe tends always to increase the first half of the phase of flight. The heavier the shoes the greater the length of the first phase of flight. Now, why? Simply because you cannot pick up a heavy weight and start off with it as quickly as you can pick up a light weight and start off with it. You cannot snatch up a pound weight and start away with it as quickly as you can snatch up an ounce weight and start away with it. Here is a horse shod with an ounce shoe. There is so little inertia in an ounce, that the muscles can take it and start away with it instantly; but here is sixteen times as much inertia or tendency to stay where it is, in a pound shoe, as in an ounce shoe, and the body has to move farther forward before the animal can start. Therefore a heavy shoe increases the first half of the phase of flight. The body is moving along at a certain rate. There is a pound shoe on that foot, and the body is moving along, and that foot gets farther back before the muscles can start it. The body is moved farther forward, and the first half of the phase of flight is lengthened. After the muscles have got it started the weight in the shoe tends to carry the shoe a little farther forward. In other words, you cannot stop sixteen ounces of weight as quick as you can stop one ounce of weight when started, and therefore a heavy shoe increases both the phases of flight and

makes the stride longer. When you get that weight started it carries the foot an inch or so forward farther than it would if it has a light shoe. It stretches things out when it gets started. It sets that foot an inch or two inches farther forward on the ground; so that a heavy shoe, or a weight in the shoe, increases the length both of the first and second halves of the phases of flight.

We have not talked about speed. The stride is longer, but it is a question whether he takes more of them to the minute or not. If he took longer steps and more of them to the minute, of course the speed would be increased, but it may be that his lengthening of the stride will slow or lessen the number of steps, and he will take a less number of steps to the minute. That has to be determined by the watch. Increasing the length of the stride does not mean an increase in speed, because he may take a less number of steps to the minute.

A member: Does it, though?

A. Yes, up to a certain point, and then the timer's watch has got to tell when that point has been reached.

The member: We take lots of horses and educate them to a fast walk by carrying weights.

Dr. Adams: That was the old method of training trotters. They wore shot bags around their ankles, to give more work for the flexor muscles of the legs, and when the muscles had been well developed by carrying weight, then they would take the weight off. During training they would increase the strength of the locomotor muscles by weighting the shoe. In colts, the weight tends to rhythm. That weight puts the foot on the ground, and when he gets it started it tends to carry it on. It is just like the fly wheel that steadies the whole machine by its weight. The time comes when the gait has been educated and the animal has that rhythm, and then the weight should be taken off.

So that is what is known as balancing, giving the feet momentum; so that if this (indicating) is a foot moving with a heavy shoe, if the animal thinks he will dwell or skip, the heavy shoe carries it down, and he does not dwell or skip. It makes for rhythm.

Q. Does not this weight give them a higher action?

A. That depends largely upon the slant of it as seen from the side. Suppose a horse has a very slanting pastern. A horse with a quite slanting pastern rolls a little higher than a horse that stands upright. The horse that has upright pasterns is apt to travel close to the ground. The foot gets farther back before he breaks over, and when it does begin to

swing up it swings through a wider circle and the foot starts out higher.

Now, take an acute angled foot (illustrating by means of a blackboard drawing). That foot breaks over slowly, but when it starts it starts high and it reaches its highest point before it passes the supporting leg, and then reaches far out and close to the ground. With the acute angled foot, the foot breaks over slowly, but when it does break over there is a sudden upward jerk, and then the weight, if he has weight, carries the foot rather high. So that weight for increasing the height of the action is most efficient in an acute angled foot with long toe and heel. The height of the stride is most efficient with a long slanting toe and low heel, and least efficient in the upright foot.

Q. Where would you place the weight?

A. The weight to increase the length of the stride or the height of the stride should always be as near as possible to the end of the toe. It ought not to be at the heel.

The member: That is contrary to custom, though, is it not?

A. Yes, it is largely contrary to custom. The center around which the phalanges rotate is practically the fetlock joint. When the weight is in the heel the radius from the center of the heel is less than when it is in the toe, and the radius is from the center of the fetlock to the toe, and the longer the radius upon the end of which the weight is placed, the more the centrifugal force and the greater the momentum, because the circle is greater. So that to produce high action and a long stride, the weight at the toe is more efficient than at any point between the toe and the center of the fetlock. The nearer the weight to the center of the fetlock, the easier it is carried by the horse, but the least bit of increase heightens the action and the length of the stride.

Q. In a trotting horse that hits his elbows, in what position would weight be more detrimental to him?

Q. Would a toe weight do any good?

A. Not as far as hitting elbows is concerned. The more you get away from weight in a horse that hits his elbows, the better you are off.

Q. A short toe would do him more good?

A. Yes, because you shorten the toe and diminish the height of the stride. With a short toe the foot starts off more nearly parallel with the ground. In a long toe it starts up.

Q. When a horse interferes, the less weight you have on the shoes the better?

A. Yes. Whenever a foot is moving in a circle, either downward or inward, shoe as light as possible. There is something we can do. You take a splay-footed horse, close between the fetlocks. Suppose you are dealing with a toe-wide, splay-footed horse close in the pasterns. That horse breaks over with the inside of the toe. Then the inside of the foot swings forward and inward until close to the supporting leg, and circles inward and upward and breaks over with the outside toe. That moves in a circle. The greater the weight in the shoe the greater the tendency to fly off on a tangent. The horse that stands in that position has a slanting outer and steep inner wall, and the longer the outer wall and the shorter the inner wall, the more toe-wide he will stand.

In dressing the toe-wide foot, spare the inner wall, shorten the outside wall. In some cases raise the inner wall by thickening the inner branch, and he will toe straighter ahead for comfort, and if you can make him travel straight ahead he will travel exactly in line. You cannot make such an animal toe straight ahead because he is not born that way. The joint does not feel comfortable that way.

Let us understand just why the foot does swing out. It is an anatomical necessity. The shoulder joint of a horse is not exactly a ball and socket joint, but it permits of a lot of rotation. The elbow joint is a pure hinge joint, like the strap hinge on a barn door. It permits extension and flexion in one plane, with no rotation possible. The carpal joint is an imperfect hinge joint, with the chief movements that of flexion. The fetlock joint is an absolutely pure hinge joint, with no rotation possible at all. The coronary joint is almost a pure hinge joint; the coffin joint is nearly a pure hinge joint; so that the only joint in the leg that permits rotation is the shoulder joint. All joints below are hinge joints.

Take an animal that is prone to stand base-wide or toe-wide. His toes point out. His elbows are quite close to his body. In order to walk, he has to shorten his leg and in order to clear the ground he has to flex the joints. His toes point forward and outward, and the axis of the joint is not transverse to the body but is at an angle with the body. When he flexes, he does it not in a plane parallel to the long axis of the body, but in a plane which is at an angle to the long axis of the body. So that when he flexes he flexes back toward the median line, and when he extends, he extends forward away from the median line. In the act of flexion he has to bring the foot close to the supporting leg, and in the act of extension he has to carry it away. If his toes were straight ahead, he would flex and extend just in a line of movement, and in that flexion and extension the foot would not pass away

from the supporting leg, but when he flexes and extends in another plane, in flexion he comes close to the supporting leg.

Q. Can a horse be shod so as to prevent interfering from leg weariness?

A. When a horse has a tendency to interfere it is always most pronounced when he is leg weary. The treatment by shoeing should be the same as when the horse interferes from any other cause. Leg weariness should be avoided if possible by less work, better feed, longer periods of rest or the purchase of better stock.

INFLUENZA AND SHIPPING FEVER

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Influenza of the Horse

Under the collective term "influenza" intermittently have been grouped together at least two acute, infectious diseases, one of which assumed the form of a general infection of the blood with inflammation of the mucous membranes, subcutis, tendon sheaths and tendons, and the other an infectious lobar pneumonia or pleuro-pneumonia. From a purely clinical standpoint some authorities (Dieckerhoff, Bang) have pronounced influenza not to be a clinical entity. They assumed that under this head at least two separate and distinct diseases existed which differed in their pathogenesis, period of incubation and symptomatology. They were therefore given separate names. In Germany the catarrhal form was known as "Staupe" (influenza) and the pectoral as "Brustseuche" ("chest plague"). Dieckerhoff described a third disease, included under the term influenza, which he called "Skalma."

The recent experiments of Gaffky and Luhrs which concerned the etiological factor primarily indicate that the term "influenza" has been made too inclusive. Their results seem to substantiate the work done by former authorities, especially Dieckerhoff and Bang. In the light of our present knowledge it seems advisable to classify under the term influenza two separate and distinct diseases: (a) Influenza. (b) Infectious fibrinous pneumonia of the horse.

Catarrhal Fever, Pink Eye, Typhoid Fever

Definition: Influenza is an acute contagio-infectious, usually enzootic, disease of the horse which is characterized by inflammation of the mucous membrane, subcutis and tendons. It is due to a filterable virus.

Occurrence: The disease is very widespread, occurring in all countries. North America was visited by an epizootic of influenza in 1776 and again in a still severer form in 1870-72, when it swept the continent from Canada to the Ohio, and westward to California. The disease is now permanent in this country, where it is kept alive by the unsanitary stables of horse dealers and in livery barns, from whence it is from time to time spread. It may also attack asses, mules and zebras.

Etiology: The most recent investigations indicate that the cause of influenza is a filterable virus which appears in the blood and probably other body fluids. Gaffky produced

influenza in healthy horses by subcutaneous injections (5 cc.) of defibrinated blood from naturally infected horses. The period of incubation is from five to six days. When the blood was injected intravenously the period of incubation was only four days. From artificially infected horses the characteristic symptoms were produced in forty hours. Similar experiments made with filtered blood serum gave positive results. The infectiousness of the blood was annihilated by the addition of the citrate of ammonia. In no case was he able to obtain growths on culture media from either the blood or blood serum used.

Natural Infection: The disease is probably spread by the nasal discharge and feces of infected horses. In all probability apparently recovered cases may be "germ carriers," introducing the disease into stables and when brought in contact with susceptible individuals. Indirectly the infection may be carried by contaminated food, bedding, manure, stable utensils, harness or in the clothing of grooms, horsemen and veterinarians. Many sale and livery stables, due to their lack of light, ventilation and cleanliness, may harbor the infection for an indefinite period ("stable miasma") and all horses, especially "green" horses from the country, placed in them fall victims of the disease. The same is true of railway cars and stock yards which have not been properly disinfected. Public watering troughs may also harbor infection and thus contribute to the spread of the disease. Although influenza is commonly enzootic it not unrarely assumes an epizootic form. While it appears at all seasons of the year, in the late winter and early spring horses seem most predisposed. This is probably due to the condition of the mucous membranes, which are generally catarrhally inflamed from "colds" in these seasons. Influenza will attack horses of all ages, but it is not so common in colts less than a year old, nor in aged horses. Horses three to five years of age are most frequently affected. One attack produces lasting immunity. Horses which have had influenza may, however, take infectious pneumonia, and vice versa. The infection is most commonly taken in with the food and water. Infection via respiratory tract is highly probable, but not proven.

Symptoms: The period of incubation is usually less than a week. A minimum period of one day and a maximum of ten days is recognized. The initial symptoms in many cases are moderately developed, but in the majority they are quite intense. Usually the first symptoms noticed by the owner or caretaker are loss of appetite, dullness and marked languor. The patient stands with head down, eyes closed, ears dropped, appearing excessively fatigued. The gait is staggering and

crackling of joints is heard when the patient is moved.

The temperature is elevated (103-105° F.). It usually drops by crisis on the second or third day. The pulse in the earlier stages is relatively low, compared with the fever, but as soon as cloudy swelling of the heart begins it goes up to 60-100 and becomes irregular and weak.

Eye Lesions: Conjunctivitis appears early and later keratitis and iritis (exudate in anterior chamber, contraction of pupil, etc.). The blood is charged with bile pigment, hence the conjunctiva assumes a yellowish, ocher or a natural mahogany color. Sometimes a phlegmonous conjunctivitis with marked swelling and eversion of the eyelids occurs. These symptoms generally rapidly subside, provided the cases take the normal benign course.

Respiratory Tract: Cough is usually present. It is generally strong and moist. Nasal discharge, at first clear, but later turbid and more profuse, is a constant symptom. The submaxillary glands are swollen. The patient shows moderate dyspnea, the respirations 20-25.

Percussion Normal: Auscultation exaggerated, vesicular breathing and moist rales.

Digestive Tract: Lost or impaired appetite and, during the fever, increased thirst are noted. Usually symptoms of catarrhal stomatitis and pharyngitis are present (coated tongue, warm, congested mucous membranes, regurgitation of water through the nostrils). The gums are swollen ("lampas") and along their border a marked yellowish discoloration is seen. The bowels during the febrile stage are constipated, the feces passed in the form of hard, small, mucous covered pellets of sour odor. Later they become soft and a fetid, exhaustive diarrhea with tenesmus sets in. In some cases moderate colic symptoms occur (pawing, lying down, slight distention of abdomen).

Urino-genital tract: During the height of the fever very little urine is voided. Its specific gravity is high, color dark, reaction acid. It is rich in sediment. Under the microscope tube-casts and epithelial cells (renal pelvis, bladder) are found in it. With the falling of the temperature a critical polyuria develops. In mares the vulva may swell and a muco-purulent discharge be present. Pregnant mares often abort. In stallions the scrotum is often enlarged and orchitis may be a symptom.

Skin: While there is generally an edema of the hind limbs present in influenza, in some outbreaks it is much more pronounced than in others. With increasing heart's weakness,

edemas appear in pendent portions of the body (under chest, abdomen, udder or scrotum). Occasional cases of tendovaginitis, particularly of the flexor tendons, are noted and once in a while laminitis occurs. The patients usually lose flesh rapidly, become very weak and debilitated.

A very dangerous complication is pneumonia or pleuropneumonia, which usually assumes the catarrhal form and may be hemorrhagic in character. Such cases seriously disturb the functions of the heart and kidneys and often lead to death.

Course: The course is usually about one week or the disease may terminate favorably in less time provided no complications arise. Where the disease progress is interrupted by pneumonia, encephalitis, enteritis or degeneration of the heart's muscle the course is protracted and the termination fatal. As a rule, however, influenza is a mild disease, and unless the patient is worked, kept in unsanitary conditions, or given too much medicinal treatment, recovery in a few days is the rule.

Prognosis: The mortality is 1-4 per cent. During some outbreaks the disease appears more malignant than in others and complications are commoner. The continuation of a rather high fever for not longer than five to six days is per se of no significance provided the pulse remains good, the patient does not become dyspneic and no diarrhea attends.

Diagnosis: The diagnosis is usually not difficult. The rapid spread of the disease from animal to animal, the icteric mucous membranes, conjunctivitis and skin edemas differentiate it from other diseases attended by high fever. Where these symptoms are absent the highly infectious character of the disease, as evidenced by its rapid spread is significant.

While a differentiation between influenza and infectious pneumonia of the horse can usually be made if the course of the disease can be studied, at first visit it may be very difficult. The prodromal symptoms of the two diseases are very similar. The high initial fever, the swelling and icteric discoloration of the conjunctiva, the contagiousness and the inflammatory swellings in the subcutis and tendon sheaths are common to both. Usually, however, on the second or third day in infectious pneumonia tangible symptoms of pneumonia develop; in influenza pneumonia occurs as a complication and usually much later in the course of the disease. Furthermore, influenza is more apt to attack the intestinal tract early. A differentiation between influenza and infectious anemia would become important only in districts where the latter disease exists. In infectious anemia no catarrhal symptoms develop,

the mucous membranes show petechiae, the blood serum is opalescent and plainly tinged with red. Infectious anemia is not highly infectious. From strangles influenza is distinguished by the tendency for lymph glands to suppurate, which characterize the former disease.

TREATMENT: Most important in the treatment of a self-limiting disease like influenza is to provide the patient with light, ventilation and cleanliness. In mild, sunny weather, if at all feasible, place the patient out of doors during the day. The food should be nourishing and easily digested and given in small rations (grass, alfalfa, a bran mash with plenty of salt, scalded oats, etc.). To induce the patient to eat, some brown sugar may be strewn over the feed. In case a meal is refused or not entirely eaten it should not remain indefinitely in the feed box, but be removed and the box cleaned. Skim milk may be given (three to four gallons daily) if the patient will drink it. Hanging blankets wet with cold formalin solution in the stable helps lower the temperature in hot weather and assists disinfection. A thorough cleaning up and disinfection of the infected quarters will do more to reduce the mortality than drugs. The unequal distribution of the surface temperature should be regulated by proper grooming. The legs should be covered with Derby or flannel bandages, which tend to prevent edema. Medicinal treatment should only be employed when absolutely necessary. Overdrugging in influenza always increases the mortality. The following conditions may require aid:

Fever: The fever should be let alone unless it reaches an unusual height or is continuing rather indefinitely and seriously affecting heart's action. It may be reduced by cold rectal infusions which not only lower temperature, but stimulate peristalsis and unload the bowels. The use of antipyretics, such as acetanilid or phenacetin, every three hours, is dangerous, as both drugs are powerful heart depressants. They should be administered, if at all, with caution. Sodium salicylate is less dangerous in this regard, but may irritate the stomach.

Heart Weakness: When the pulse becomes rapid and weak one dose of digitalis (Squibb's fluid extract) has a remarkable toning effect. Strophanthus, camphor oil subcutaneously are also effective. When the pulse is strong enough, alcohol with ether in a pint of cold water is of service.

Gastro-intestinal Tract: Artificial Carlsbad salts, to which bitter agents, gentian or nux vomica are added, are useful. Dram doses of a solution of strychnine nitrate in water may be used. Bowel disinfectants are also employed, as creolin

or naphtholin. Diarrhea may be fought with astringents like tannin with opium. Starch clysters are helpful. The profuse diarrhea attending septicemia is impossible to check. Calomel with bicarbonate of soda mixed with powdered licorice root, smeared over the teeth as an electuary, may be tried.

Skin: Leg swellings are best reduced by elastic bandages, and if the pulse will permit, moderate exercise. Burrow's solution to which a little gum camphor is added may be used as a leg wash under the bandage.

The eye should be treated as in conjunctivitis (boric acid solution, 2%) and the pulmonary and pleural symptoms met as recommended under the diseases of the respiratory tract.

Convalescent patients should not be worked until strong enough and full appetite returned. The animal is generally fully recovered fourteen days after the temperature has become normal.

Infectious Fibrinous Pneumonia of the Horse (Chest Plague, Pectoral Influenza, Pleurisy, Shipping Fever)

Definition: An acute, febrile, contagio-infectious disease of the horse which in typical cases appears as a fibrinous pneumonia or pleuro-pneumonia with which is associated inflammation of the subcutis and tendon sheaths. In mild cases it may assume the form of a general febrile disease of short duration.

Occurrence: Infectious fibrinous pneumonia is widespread, occurring in all countries. Statistically it is difficult to state, however, anything definite in regard to its prevalency, as it has been so often confused with influenza. The disease is most apt to appear in large stables where many horses are kept, and especially during inclement weather. The disease in passing through a stable does not usually affect the horses in the order in which they stand in the stalls, the infection tending to spread unevenly, skipping apparently susceptible horses. Infectious pneumonia does not become epizootic as readily as does influenza; it is more apt to remain confined to an infected stable, from which it may be spread, however, by a convalescent horse. While infectious pneumonia will not involve in a given country as many horses as influenza, on account of its higher mortality and a greater tendency to leave behind sequelae ("heaves," relapsed cases) which may permanently impair the efficiency of the horses attacked, it can assume even greater economic importance.

Etiology: The cause of infectious pneumonia of the horse has not yet been identified. According to Gaffky and Luhrs the bronchial slime of an infected patient contains the virus of

the disease, at least in the early stages. In typical cases of infectious pneumonia, where the patients were destroyed on the third or fourth day, in the bronchi was found a quantity of yellow, transparent, viscoid fluid which contained no bacteria. In two experiments on twenty-four colts inoculated with this fluid, by painting it upon the mucosa of the nostrils and in the mouth, the colts became typically ill with infectious pneumonia in twenty-three to forty-two days. Undoubtedly some specific virus, which may be filterable, forms the true cause of the disease. In all probability, however, other bacteria, principally the streptococcus, pyogenes equi and the bacillus equi septicus, are secondary invaders contributing to the underlying disease process and clinical phenomena.

Natural Infection: The way in which the disease spreads naturally is at present not known. The infection seems to be spread by more or less intimate contact between the sick horse and susceptible ones. The transmission through intermediate agents such as food, water, stable utensils, etc., or through persons or insects has not been demonstrated experimentally. However, practical experience in other diseases and with this disease would not exclude indirect transmission. Sporadic outbreaks in stables could be explained, however, as coming from apparently healthy "germ carriers" or "missed cases" of infectious pneumonia, i. e., where still exist in the lungs or occasionally in other organs unhealed foci of infection. As predisposing factors anything which reduces the resistance of the susceptible animal unquestionably has a bearing on the origin of the disease. Therefore refrigeration, overwork, bad sanitary conditions, become predisposing factors. The disease is rare in very young or aged horses and occurs usually in animals in the prime of life.

One attack produces immunity for only a short period. Individual instances are recorded where a given horse has suffered repeated mild attacks of the disease. An attack of infectious pneumonia in no way influences the susceptibility of influenza infection.

Necropsy: On post portem, where the disease has assumed a typical form, the lesions are those identified with fibrinous or even hemorrhagic pneumonia with a marked tendency to gangrene. The extent and distribution of the inflammation varies. In some cases the pneumonia is of the lobar, in others the lobular type. The exudate is usually of a hemorrhagic character. Yellow-gray areas of necrosis throughout the lung tissue are commonly noted appearing as encapsuled pus centers or gangrenous foci.

The pleuritis is sero-fibrinous; the thorax may contain several gallons of serous exudate. Adhesions between the lung

and chest wall (adhesive pleuritis) is a common finding. Empyema of the thorax may be present.

In the gastro-intestinal tract appears evidence of catarrh and, especially in the small intestines and cecum, inflammatory thickenings and ulcerations.

Symptoms: Infectious pneumonia of the horse is characterized by pneumonia and pleuritis. The pneumonia may assume either the lobar (benign) or lobular (malignant) type.

Complications: The most dangerous complications are: (a) Parenchymatous degeneration of the heart characterized by tumultuous heart beat and a rapid, arhythmic, weak pulse. (b) Nephritis distinguished by scantiness of urine (anuria) and albuminuria. (c) Septicemia, usually following pulmonary gangrene, and recognized by continued high fever, chills, rapid, irregular and weak pulse and exhaustive diarrhea. (d) Paraplegia is rarely noted, but is usually a fatal complication. (e) Paralysis of peripheral nerves (facial, recurrent) which may persist for several weeks. (f) Swellings of the limbs and pendant parts of the body, seen especially in the latter stages, are troublesome. (g) Tendinitis and tendovaginitis. (h) Founder. (i) Abortion. (j) Decubitus.

Diagnosis: Usually not difficult. Infectious pneumonia cannot be differentiated from sporadic lobar pneumonia, if such occur in the horse. Every case of lobar pneumonia, therefore, should be isolated. Obviously this does not apply to all lobular pneumonias, especially foreign body, medicinal or hypostatic.

Course: When the pneumonia assumes the lobar type with mild pleuritis, the course is about two weeks. If, however, the lobular form of pneumonia with pronounced bilateral pleuritis is present the course is prolonged for weeks, with remissions and exacerbations, either ending finally in death or leaving the patient permanently windbroken (pleural adhesions, roaring).

Prognosis: The mortality is 1-4 per cent. The following factors are important in the prognosis; they are all grave symptoms: (a) Heart paralysis, rapid, empty pulse, venous pulse; (b) extent of pneumonia and pleuritis, bilateral with great effusion; (c) pneumonia centralis, sudden dyspnea with rise in temperature during course of the disease; (d) hemorrhages, epistaxis, bloody pleural exudate; (e) temperature continues high for over a week or is remittent in character; (f) diarrhea; (g) lung gangrene; (h) brain symptoms.

METHODS OF SECURING AND SHIPPING TISSUES FOR LABORATORY EXAMINATION

DR. L. E. WILLEY, AMES, IOWA

The increasing demand by veterinarians and stock owners for assistance in investigating obscure cases of infectious and non-infectious diseases of animals has necessitated special attention which the Department of Veterinary Research and Investigation of the Iowa State College is attempting to supply.

Numerous cases on which much time, effort and material were expended last year resulted in negative findings, so far as diagnosis is concerned on account of specimens being received in such an advanced state of decomposition that satisfactory examination was impossible.

In other cases material would be sent to the laboratory in such a strong preservative solution with the request to look for a particular organism. It is evident that animal inoculation or cultural methods are out of the question.

Many specimens were received without the name of the shipper and unaccompanied by letter stating what the specimen was and what sort of an examination was desired. It seems that a description of some of the practical methods of securing and shipping tissues to the laboratory should be considered here.

Autopsy: Object of.—The object of an autopsy is to diagnose the cause of the animal's death or to confirm the clinical diagnosis.

Time: This depends on circumstances of the case and may vary from a few minutes to a longer period of time.

In most instances it should be performed as soon after death as possible.

In some cases, especially septicemias, it appears to be of some assistance, at least no harm, to permit the carcass to remain undisturbed for a short time (two to three hours), during which time organisms will multiply to the extent that they are more readily found.

The cases in mind applying to this statement are cases of hemorrhagic septicemia in which, being called upon to make examinations, and upon drawing the blood from vessels or the heart, no organisms were found on microscopical examination.

However, the organisms later were demonstrated on animal inoculation. Sometimes this consumed a considerable pe-

riod of time. Other cases where examinations were requested, and which had been dead for three, six or more hours, organisms were found very abundantly in the blood and other fluids of the body. These are the special cases in which a delay of the post mortem sometimes facilitates the discovery of the etiological factor.

Should the lesions found be atypical or insufficient for a field diagnosis or a confirmation of the field diagnosis is desired, it will require one or more of the methods applicable in an equipped laboratory.

For the assistance and guidance of the men who examine the material in the laboratory, it is highly important that a simple system of examination in the field be followed, for a post mortem on a particular case does not admit of repetition.

If, in performing a post-mortem examination on an animal suspected of having died of hemorrhagic septicemia, the carcass is carelessly opened, cutting into the intestines or the stomach, and getting that fluid on the knife, and then opening into the thoracic cavity and slicing into the heart, when a hemorrhage is suddenly discovered and then an attempt to collect that blood and send it to the laboratory for examination, and upon inoculation of experimental animal and that animal dies within twelve hours with pronounced lesions of emphysema, the case is complicated, and an erroneous diagnosis may be rendered, whereas, if care of knives and the way the post mortem is performed, all of this difficulty may be avoided and the mistake in the laboratory diagnosis may be avoided as well.

This report should in every instance accompany the specimen. It should include clinical examination of the subject previous to death if possible.

1. Inspection of the exterior of body.
2. Topographic exploration abdominal cavities and organs.
3. Topographic exploration thoracic cavities and organs, after which detailed examination, including removal and incisions, may be made.
    1. Situation and relations of organs.
    2. Size and weight.
    3. Shape, contour, border, coverings.
    4. Color.
    5. Consistency.
    6. Anomalies and malformations.
    7. Fractures, dislocations, lacerations.
    8. Cut surfaces and liquid exuded.
    9. Odor.

10. New growths.

11. Pathologic conditions.

Note taking important.

Memory is often treacherous.

When writing your report not to make it voluminous or irksome, negative lesions may not be noted unless important. In the failure to note lesions, their absence is assumed.

Preparation for Autopsy: This includes adequate instruments and protection to the operator by proper clothing if the disease is communicable.

In particular cases the veterinarian should be prepared to properly collect such tissues as may be necessary for further study in the laboratory.

Preparation of Tissues: It is necessary for the veterinarian to decide the kind of examination desired. Experience seems to justify a rather definite description of satisfactory methods of collecting tissues and specimens and preserving the same for transportation to the laboratory.

Gross specimens for macroscopic examination or museum purposes may be treated and packed according to the nature of the tissue.

Large specimens of such size as to be impractical to send in fluid preservative may be immersed in a solution of formalin, 10 per cent; i. e., one part formalin to ten parts of water for twelve hours, drained, wrapped in several thicknesses of paper, then boxed for shipment. Should the specimen contain much liquid, absorbent material should be added.

Small pieces of more delicate tissues, including tumors and tissues showing petechial hemorrhages, etc., are best shipped in a solution of formalin not stronger than 50 cc. formalin to 1,000 cc. water (2 per cent formaldehyde or 5 per cent formalin).

Formalin solutions in these strengths will sterilize tissues if left long enough in contact with them, but if not left too long will not bleach the tissues to the extent that hemorrhages and exudates will be unrecognizable.

On no account use formalin in full strength for preserving tissues. It will effectively bleach and harden tissues in a few hours, rendering hemorrhages and exudates unrecognizable.

Another medium which is quite satisfactory is glycerine 50 per cent, water 50 per cent, even when pure does not sterilize tissues for some time.

Alcoholic solutions are least satisfactory, but when necessarily used should be diluted with water to 70 to 80 per cent.

Tissues for Bacteriological Examination: Blood.—This is

most satisfactorily collected in small bottles previously sterilized by steam or boiling water.

A sterile hypodermic syringe and long needle will be of great aid in securing the fluid free from outside contamination.

It is best drawn from the jugular after skin has been carefully removed or from the heart after removing the sternum. Other body fluids are collected in a similar manner.

Pieces of tissue in addition are most satisfactorily packed in a sterile friction top can, such as an empty syrup can or pail, or fruit jar.

The vessel can be packed in a box surrounded with cracked ice and sawdust. The sawdust prevents rapid melting of ice and will preserve tissues for twenty-four to forty-eight hours or longer if properly done.

Sections of intestines and vessels are best ligated before removal from the body.

Care should be exercised to keep intestines separate from contact with other pieces of tissue, as the bacterial flora in them causes rapid decomposition and often obscures or complicates examination of other tissues.

A year ago, following the method of shipping fresh tissues to the laboratory as outlined here, specimens were received from a small town in Pennsylvania. Directions were sent to those people to pack the material in an ordinary quart syrup can, and to solder the top. Then to put two slats across the inside of the box to hold the can in place, and to pack around the can sawdust and cracked ice, and that material reached the laboratory in satisfactory condition for bacteriological examination.

Specimens were prepared yesterday to illustrate the action of these preservative solutions on the tissues. (Exhibiting same.) A piece of heart muscle and spleen were put into pure formalin. It is noticed to have a gray appearance. Frequently specimens sent to the laboratory have been placed in pure formalin for perhaps forty-eight or seventy-two hours, and the appearance of the tissues when it reaches the laboratory can be appreciated. If it happens to be a lung, hemorrhages or exudates cannot be distinguished. The lung still shows an area giving some indication of an appearance of hemorrhage, but that is quite exceptional. That illustrates the condition in which the tissues sometimes reach the laboratory.

Contrast the specimen now being shown in 2 per cent formaldehyde (or 5 per cent formalin). The pink appearance of the lung can still be distinguished (exhibiting same). This kidney (referring to same) still shows distinct petechial hem-

orrhages. It probably cannot be seen from where you sit, but if examined petechial hemorrhages on the surface of the kidney will be observed. That was preserved in a 5 per cent solution of formaldehyde and the difference in the appearance can be seen (referring to another specimen). This specimen is preserved in 50 per cent glycerine and water, which effectively retards decomposition. The color is still pink, and the hemorrhages are very distinct. A larynx still shows the delicate petechial hemorrhages which were observed when it was first taken from the animal (exhibiting same). Those are very delicate, and that is a hard piece of tissue to preserve.

The kidney is somewhat darkened, but still shows the hemorrhages. It does not blanch the tissues like the pure formalin.

In addition to that, when sending in lymph glands for bacteriological examination, sometimes one is so situated that it is impossible to get formalin. A piece of cotton, or gauze may be taken preferably, and wrap the tissue in it and pour on a small amount of 50 per cent glycerin and water. The lesions are very distinct. The color is not destroyed. From such a specimen as that, from the spleen especially, when it gets to the laboratory, if it happens to be a case of bacterial infection, by removing the outer surface, laboratory workers are able to take some of that tissue and grind it up with salt solution and inoculate a guinea pig, and if it contains the organism for which the examination is being made, it will undoubtedly be found. Even pure glycerine will not sterilize the tissues for some considerable length of time. Are there any questions?

Q. The glycerine and water is all right where you want to make animal inoculations, is it?

A. Yes, it is all right if you cannot get ice. Of course, we would much prefer to have the tissues reach here in a fresh condition, in which no preservatives of any kind have been used.

Q. You simply put your tissues in a pail and pack ice around the outside?

A. Yes, put the tissues in a pail, using care, as I said, not to contaminate the tissues in which you expect the organism to be found, with intestinal contents.

Q. How would water and alcohol do?

A. That is to me the least satisfactory, but when you are out in the country—now, I am speaking as much from a practical standpoint as possible—when you are in the country and run up against a piece of tissue, if it happens to be a tumor or some other piece of tissue, on which you want an examination made, of course if you have not anything but alcohol, use

it; but you generally can carry it to your office and secure something else. If you have to use alcohol, dilute it with 25 or 30 per cent of water. Do not use pure alcohol. Once in a while, in a very large piece of tissue, which reaches us in a short time, which has been preserved in 5 per cent formaldehyde, we are able to get into the interior of the piece of tissue and successfully cultivate any micro-organisms which may be there, and reproduce the disease by animal inoculation.

Q. How would salt water be for a few hours?

A. Salt water is not very satisfactory. We have received a few pieces of tissue packed in dry salt. In some cases that would be all right. For instance, I received a piece of tissue from Mexico, a piece of muscle from a hog, packed in salt, and upon soaking and washing it we were able to demonstrate the cysticercus cellulosum. It is not very satisfactory, except for parasites or something like that.

POISONOUS PLANTS

DR. L. H. PAMMEL, AMES, IOWA

Mr. Chairman and Gentlemen of the Practitioners' Course: I am pleased to address you on the subject announced in your program, because it is a genuine pleasure to meet the veterinarians. You represent a profession that is indispensable to the welfare of our commonwealth. The time was that your profession did not have the standing it has today. It is largely through the advance of exact science that your profession ranks with medicine and pharmacy in the permanent upbuilding of the commonwealth. The discoveries that have been made by the veterinary profession have added millions to the wealth of our country.

In a discussion of poisonous plants I shall confine myself largely to Iowa problems and if the editors of the admirable brochure recently issued by the Iowa State College, Veterinary Department, deem this paper worthy of publication you may have access to a list of the more important poisonous plants of the state. Let it be understood that I place the widest interpretation on what constitutes a poisonous plant.

Kobert and other physicians define a poison as "a non-organized body, either organic or inorganic, which under certain conditions, affects temporarily or permanently one or more organs of the body, when in a state of health or in a healthy condition." Such poisons may develop in the body or may come from without. Some substances act injuriously in a mechanical way, that is, they may set up disturbances by irritating some parts of the body. Other substances, while poisonous to one who is ill, may be entirely harmless to persons or animals in a state of health.

The subject of toxicology still holds a prominent place in law, medicine and veterinary practice. However, the botanical treatises on poisonous plants are not as numerous as they were fifty or seventy-five years ago. Our knowledge in regard to the action of poisons is made more exact today than it was when Buchner, Orfila, Plenck, Fordore, Taylor, Ratzeburg, Sorbernhein and Simon wrote their respective treatises.

In a study of poisonous plants we must consider two factors: First, the nature of the toxic substance; and second, the individual. The snail is said to be capable of withstanding more strychnine than an adult man.

Cardiac poisons produce no action upon insects. The rabbit can stand more morphine than a man. Kobert says: *"Amyg-*

dalin does not affect dogs, but it kills rabbits. The hedgehog takes, with apparent enjoyment, a dose of *cantharides* that would kill several persons under excruciating pains. The bite of the most venomous snake does not harm him; he can even accommodate no inconsiderable quantity of *hydrocyanic acid.* Whereas the frog is extraordinarily susceptible to the *digitalis* poisons, they have no effect on the toad. Poisons act more powerfully when absorbed from the subcutaneous connective tissue than when administered internally, with the following exceptions: The neutral *crotonologlycerid,* which is found in large quantities in the fresh seeds of *Croton Tiglium,* but which is often lacking in commercial croton oil, is inactive when introduced under the skin. It possesses, however, terriffic action when taken into the stomach. *Myronic acid* of mustard as an alkaline salt has no effect when it is injected under the skin; it has, on the other hand, a strong action when taken *per os* by herbivora; the same is true of *amygdalin.*

"In all three of the foregoing cases, the apparent exception to the rule is explained by the fact that the substance, in itself not poisonous, is split up in the intestinal tract, giving off, amongst others, a toxic substance. In the instance first mentioned, *crotonilic acid* is the poison thus freed; in the second, etheral *mustard oil;* and in the third, *hydrocyanic acid.* Some substances, such as *salts* of *manganese, iron, tungsten,* have no poisonous action when introduced into the intestinal tract, because under these conditions only very minute quantities are absorbed; others are rendered inert because they are excreted almost as quickly as they are taken up, curare being an example; and yet others, such as *snake poison, spider poison, quillaic acid, sapotoxin, ergotinic acid,* are converted into non-poisonous substances within the intestines." Let us take up some specific poisonous plants.

Bacteria: I need not dwell on the poisons produced by bacteria since this has or will be touched on by others.

The bacterial poisons are produced by two classes of organisms. First, the poisons produced by pathogenic organisms, like the tetanus bacillus and diphtheria bacillus. Second, poisons produced by saprophytic species. In this category we have the organisms found in putrid material, fermenting substances, mouldy fodder, etc. Not all of these organisms, however, produce poisonous substances. The type of toxic action may be illustrated by the disease known as *Botulism.*

This belongs to the first group and is caused by the *Bacillus botulinus,* which contaminates ham, sausage and fish, and is so poisonous that it frequently is the cause of death in the person using the affected meat or fish. Rabbits, guinea pigs and cats are very sensitive to the poison and die when given the fluid culture, rabbits succumbing in thirty-six to forty-eight hours after

injection of 0.003-0.001 c. c. *Proteus vulgaris,* growing in pork and beef sausage, produces a similar poisoning.

Symptoms: "Epigastric discomfort, belching nausea, vomiting gripes, diarrhea followed by constipation;" nervous symptoms appear in a few days; these are dilated pupils, paralysis of the tongue and pharynx, loss of voice; death may follow delirium and coma, or, after some time, recovery may take place.

Poisoning from this cause manifests itself within a half hour occasionally, generally within twenty-four hours, although it may be delayed a week.

Algae: The algae are humble, green microscopic plants generally found in water. The green slime *(Spirogyra)* found in water is a common type found in running water which is not injurious. The type of algae found in water which are not strictly poisonous, but decidedly objectionable, are the Blue Green Algae. One type occurs in watering troughs. It forms blue-green filaments. It is not poisonous, but is decidedly objectionable. This growth is easily prevented by washing the tank with a weak solution of iron sulphate. Other types of blue-green algae in our fresh water lakes are *Rivularia, Anabaena* and *Clathrocystis.* These float in water and present the appearance of fine granules floating in the water. By their decomposition these algae produce pig pen odors so characteristic of fresh water lakes. The floating of these algae is often referred to as "the working of the lakes." So far as I know, these algae are not directly poisonous, but such water is not very wholesome. Some of the blue-green algae are reported poisonous. We have none of the strictly poisonous species in Iowa.

Fungi: Fungi do not contain chlorophyll. They are not differentiated into root, stem and leaf, but consist of a plant body. Fungi are unable to make their own food and hence must get it from dead organic matter *(saprophytes)* or from some living organism *(parasites).* The saprophytes are represented by the toadstool and mushroom. The parasites live either on plants or animals. The favus fungus lives on animals, while ergot and rust live on plants. Just a few words about fleshy fungi. Some of the fleshy fungi are powerfully toxic. Among these is the Death cup *(Amanita phalloides),* which occasionally occurs in Iowa. The fungus has a whitish cap and a bulb. The fungus dissolves the red blood corpuscles, hence its action is much slower than the Fly Agaric *(Amanita muscaria).* This fungus has a red cap covered with whitish scales. This fungus contains the substance, muscarine. The symptoms of poisoning are as follows:

The symptoms of poisoning from the fly amanita, as deduced from a number of cases, are varied. In some instances they begin only after several hours, but usually in from one-half to one or two hours. Vomiting and diarrhea almost always occur, with

a pronounced flow of saliva, suppression of the urine, and various cerebral phenomena beginning with giddiness, loss of confidence in one's ability to make ordinary movements, and derangement of vision. This is succeeded by stupor, cold sweats, and a very marked weakening of the heart's action. In case of rapid recovery the stupor is short and usually marked with mild delirium. In fatal cases the stupor continues from one to three days and death at last ensues from the gradual weakening and final stoppage of the heart's action.

I have received from veterinarians numerous specimens of supposed poisoning from these fleshy fungi. The specimens sent, however, were of the harmless kind. We may conclude that live stock is rarely poisoned by these fungi.

Ergot: This parasitic fungus is rather common on our wild grasses, wild rye, quack grass, also on cultivated grasses like timothy, red top, wheat, rye, occasionally on oats and barley.

The disease is known as ergotism and occurs in bovines and is caused by the ingestion of considerable quantities of food contaminated with ergot. Equines are apparently less susceptible than bovines, although the horses have been known to suffer severely from the disease. Ergotism in man is not an uncommon occurrence, and in nearly every instance it has resulted from eating bread made of ergotized grain. The disease makes its appearance among cattle chiefly in the winter and spring seasons and has at times been the cause of serious losses throughout the central and western states.

Ergot is the sclerotium of a parasitic fungus, *Claviceps purpurea,* which infests many species of native and cultivated grasses and appears on some of our grains, especially rye. The sclerotium represents a stage in the life history of the fungus which is intermediate between that of the mycelium or spawn, and that of the spore-bearing thallus. It flourishes particularly well on rich soil and in warm, damp seasons. The chemistry of ergot is not definitely known, although Kobert succeeded in separating three bodies, namely, ergotinic acid, cornutin and sphacelinic acid.

Symptoms: Ergotism manifests itself among animals chiefly in the chronic form, since, as a rule, the poison is acquired in small amounts and accumulation takes place slowly. Two distinct types of the disease are recognized, namely, spasmodic and gangrenous. Symptoms referable to the digestive tract, such as nausea, vomiting, colic, diarrhea or constipation appear in both forms. Pregnant animals very frequently abort.

In the spasmodic type of the disease, symptoms due to overstimulation of the central nervous system appear. These are tonic contraction of the flexor tendons of the limbs, anaesthesia of the extremities, muscular trembling, general tetanic spasm,

convulsions and delirium. Death usually occurs from secondary causes.

Gangrenous ergotism is attributed to prolonged constriction of the arterioles, and more directly, perhaps, to degenerative changes in the vessel walls, and the consequnt formation of hyaline thrombi.

It is characterized by coldness and anaesthesia of the extremities, followed ultimately by dry gangrene of these parts. The effects of this dry gangrene are often very serious and amount to sloughing of the feet, tips of the ears, tip of the tail, shedding of the hair, teeth, etc. Death takes place from exhaustion.

Lesions: With the exception of the gangrene which may vary greatly in the sensory area of the cord and in the vessel walls have been observed in animals slowly poisoned with ergot.

Monascus: In recent years several outbreaks have been reported in Iowa of poisoning from silage. The animals are gaunt, depressed, have stiffness of gait and are unable to swallow. An early symptom, Dr. Stange says in the cases observed by him, is the ptyalism, due to inability to swallow. This mouldy corn silage is reddish in color. A number of moulds have been found, one of the most abundant being *Monascus.* It is probable that the *Monascus* secretes an enzyme which is injurious.

We have a number of other common moulds in Iowa. Mouldy hay frequently contains the common *Aspergillus glaucus,* which is certainly not pathogenic, though some writers claim it secretes a poisonous substance.

We also have in Iowa two other species of *Aspergillus.* The A. fumigatus, a fungus resembling the *A. glaucus,* but with a smoky color. The *A. niger,* another closely related fungus, is of a blackish color. Both of these species are pathogenic. The most frequent cases of aspergillosis in birds are caused by the *A. fumigatus.*

The common Mucors or Black Moulds, widely distributed in Iowa, we now know produce poisoning when the macerated material is injected intravenously.

Fusarium: During the last few years we have had a great deal of trouble in Iowa with a fungus known as *Fusarium.* There are several species; they produce a pinkish or whitish color. These moulds not only occur on the ears of corn, but cornstalks. I am inclined to think that these moulds are toxic.

Equisetosis: In Iowa, as in many other sections of northern United States, there are several species of Horsetails *(Equisetum)* that are known from direct experiment to be injurious. The disease it produces is known as *Equisetosis.* It is a rush-like plant easily separated at the joints. The dried plant alone seems to be poisonous.

Symptoms: The effects of poisoning from eating horsetail appear at times varying from two to five weeks, depending upon the age of the animal, and the amount of contaminated hay ingested.

The first symptoms are usually unthriftiness, general bodily weakness and emaciation. The animal seems to have a depraved appetite, preferring the plant to wholesome food. As the disease progresses the muscular weakness becomes more pronounced, the animal loses muscular control and exhibits incoordinate movements. During this stage the pulse and temperature are depressed, extremities are cold and the visible mucosae are pale. Appetite usually remains good until the end and consciousness is apparently retained. Finally the animal falls, manifests nervous excitement, paroxysms of convulsions appear and death results from exhaustion. In the final stage the pulse becomes accelerated and the temperature elevated.

Larkspur Poisoning: The larkspurs *(Delphinium)* belong to the crowfoot family. Many of the plants of this family are acrid, among them the Cursed Crowfoot *(Ranunculus sceleratus)*. They cause blisters to form. We have only one species of larkspur common in Iowa, the White Larkspur *(Delphinium carolinianum)*. It occurs on gravel knolls or sandy soil.

Symptoms: In domestic animals the symptoms are debility of the rear and lower extremities, producing motor paralysis. In lower animals the normal functions of the larynx become impaired because of paralysis.

The aconites or Monkshoods are very poisonous, but we have only one species in Iowa, found in Allamakee county. The effects of poisoning are tingling sensation followed by a burning sensation, reducing of the pulse, which also becomes weak, irregular and intermittent. Death is caused by stoppage of respiration.

Lupine: Fortunately, we do not have much of the injurious lupine in the state. Nor is the lupine cultivated here as in Europe. The disease, lupinosis, occurs either in the acute or chronic form. In the acute form there is loss of appetite, fever, hurried breathing, rapid pulse. Icterus, which may be detected in the conjunctiva or urine, usually appears on the second or third day.

Poisoning From Sorghum: Sorghum is a most excellent forage plant and much planted in Iowa. It should, however, be pastured sparingly. When, however, sorghum is thoroughly cured it makes an excellent forage plant.

The substance *dhurrin* C_{14} H_{17} NO_7 occurs, according to Dunstan and Henry, in young plants of *A. Sorghum.* A glucoside resembling that found in almonds also occurs; it differs, however, but is capable of being converted into hydrocyanic acid,

HCN. The investigations made at the Nebraska station show that the prussic acid is not present as such, but that it is liberated from a glucoside (1) by an enzyme in the plant, as in the case of sorghum poisoning, and (2) by the action of boiling water on the plant. Glucosides of this sort are in themselves harmless and are dangerous only when they liberate prussic acid. The experiments mentioned above also showed that even dried plants may contain a very large amount of combined prussic acid. We should expect that such a fodder would be as fatal to stock after curing as when standing in the field. Experience, however, seems to prove the contrary. Enzymes rapidly become inactive when dried in the presence of protein substances, according to Dr. A. F. Woods, director of the Minnesota Experiment Station.

Darnel is another poisonous grass. This is not uncommon in oat fields. The grass somewhat resembles quack grass. The poison is a narcotic principle. The effects are similar to that produced by sleepy grass *(Stipa robusta)*.

It is thought that a fungus found in the fruit of the Darnel causes the poisoning.

Several grasses in Iowa produce mechanical injuries like the squirrel tail grass *(Hordeum jubatum)*, which is common everywhere in Iowa. The awns of this grass work in under the gums, cause inflammation and finally the teeth drop out. In recent years another grass, the Awned Brome Grass has become common in Iowa.

This plant produces injuries similar to those caused by squirrel tail grass, the awned glumes working in under the teeth, causing inflammation and suppuration. Animals eating this grass may lose their teeth as a consequence.

Wild Cherries: We have two species of wild cherries in Iowa, the Choke Cherry *(Prunus Virginiana)* and the Black or Run Cherry *(Prunus serotina)*. Other species of the genus *Prunus* produce prussic acid. These plants all contain the bitter glucoside, Amygdalin, and an enzyme known as Emulsin, which in the presence of water acts on the glucoside and converts it into hydrocyanic acid.

The symptoms of poisoning are as follows:

In poisoning, the blood becomes first a bright arterial hue, and later assumes a dark, venous color. The first condition is due to the fact that the blood does not give up its oxygen for some reason. Brunton suggests that it is because the blood is hurried so rapidly through the dilated peripheral vessels that it does not have time to yield up its oxygen. The dark color of the blood is probably owing to asphyxia and accumulation of carbonic dioxide, following the paralytic action of prussic acid upon the respiratory center.

Prussic acid has an essentially depressing action upon the nervous system as a whole. The brain, cord and nerves become paralyzed by large doses.

The spinal cord is paralyzed at a period after coma and convulsions have appeared. The peripheral nerves and muscles are paralyzed directly by toxic doses and not through the mediation of the central nervous apparatus.

Inhalation of the pure acid will cause death in a confined atmosphere and even inhalation of the medicinal solution will induce the physiological symptoms of the drug.

Fresh leaves are generally considered harmless, but Chesnut says that cattle are frequently poisoned from eating the wilted leaves. He also adds that the seeds of all varieties of cherries and plums, both native and introduced, are subject to suspicion. The flesh of none of the species is in any way poisonous. Chesnut also says in another contribution that no cases are on record where stock have been poisoned by eating the leaves of any species while still on the tree. It is only after they have been cut off and are partially wilted that they are considered dangerous. The reason for this is that during the process of wilting, prussic acid is formed from non-poisonous constituents which are always present in the leaf and bark. The wilted leaves have the characteristic odor of prussic acid.

Rattlebox: In the Missouri river bottom, especially on sandy soils, there occurs a leguminous plant with yellow flowers and later a greenish pod becoming black known as the Rattlebox *(Crotalaria sagittalis)*. Many horses have been killed by it.

The disease in most cases is very slow in its progress, but proves almost uniformly fatal after a number of weeks or months. There is a general decline of bodily vigor throughout this period, and the only abnormal symptom in many cases is that of marked emaciation and consequent weakness. Horses that have been kept at pasture through the summer, without work, and where the grass grew in greatest abundance, were so thin in flesh that they walked with the greatest difficulty. A critical examination of many of these patients revealed nothing more than the condition resulting from starvation. This was not uniformly the case. In a number of instances there was marked coma or stupor, the animal often falling asleep while eating. In some instances the animal would remain standing for a whole week, sleeping much of the time with head resting against some object. In a few instances the animal lost consciousness and broke through fences and other obstructions.

Stemless Loco Weed: Stock poisoning by the loco weed is a frequent and serious condition with which the stock owners of the western half of the United States have to contend. Mon-

tana and Colorado, especially, sustain heavy annual losses. Similar diseases occur in other parts of the world. In Australia other plants of the order *Leguminosae,* like *Gastrolobium,* produce similar symptoms. Maiden states that the "Nenta Lessertia disease of South Africa is identical with a disease of the pea-eating animals of Australia and with the loco disease of the United States." Many forage plants of excellent repute, such as white clover, alfalfa, lotus and other plants, may produce tympanites.

Symptoms: Difficult breathing; the poison enters the circulation and stops the action of the lungs and heart, when the animals stagger and die.

In the advanced stages the animals become frantic, hence the name "loco" or crazy. Horses and sheep are the most susceptible, although cattle are also affected.

Of the various species of loco weed, the stemless loco *(Oxytropic Lamberti* Pursh), and the woolly loco weed *(Astragalus mollisismus* Torr), are the most injurious. These weeds grow luxuriantly on sandy ranges and appear early in the spring when other vegetation is scarce, and since they retain their fresh green color during the entire summer they prove especially attractive to stock.

Recently the poisoning has been attributed by Dr. Crawford and others of the U. S. Department of Agriculture to mineral salts in the plant. The period of greatest danger is chiefly during the month of May.

Symptoms: The symptoms, which are referable to the nervous system, are attributable to the narcotic effect of the plant. They appear slowly and are apparently divisible into two stages. The first stage is characterized by the following symptoms: Stupor, defective vision, unnatural movements and apparent hallucinations. When excited the animals become frenzied. The coat becomes shaggy, the teeth grow long and become loose, and a depraved appetite, which is very marked, is developed. The animals prefer the loco weed to wholesome food and will dig up the roots and eat them to satisfy their craving.

In the second stage there is emaciation, exhaustion, feeble movements and finally death from starvation. The course of the disease is quite variable and may last from a few months to one or two years. Sheep manifest symptoms very similar to those above described.

Black Locust: The Black Locust, which is commonly planted as an ornamental tree, has in a number of instances caused death. Dr. H. S. Murphey also reports poisoning to a gray mare which ate some of the bark of this tree. Dr. Murphey describes the symptoms as follows:

1. Animal stands back from the manger with legs well apart,

resting nose and mouth on floor most of the time; a part of the time head is held eighteen to twenty-four inches from the floor.

2. A continual "thud" is heard, which is not synchronous with respiration, but is synchronous with a vibration of thorax.

3. Temperature, 99.C Fahr.

4. Pulse, 54, full, bounding.

5. Respiration, 14, very feeble.

Poisoning of Man and Animals From Cowbane

Cowbane (Cicuta maculata) are not infrequent.

The Cowbane *(Cicuta maculata)* and related species belong to the Carrot family. The Cowbane is common in low grounds in the northern states and in the Rocky Mountains; another species *(C. vagans)* occurs on the Pacific coast, and a third species *(C. bulbifera)* is a common bog plant in the northern states.

The Cowbane is frequently miscalled Wild Parsnip. The latter, however, has a conical root and is the feral form of our cultivated species, and although it may be somewhat injurious, as indicated elsewhere, is not poisonous like the Cowbane *(Cicuta maculata).*

Cowbane is a smooth, marsh perennial plant from two to five feet high, with fleshy, fascicled roots and a pungent odor; leaves are pinnately compound with coarsely serrate leaflets; flowers are white and small; fruit broadly ovate to oval and small. The European species has long been recognized as poisonous. Many cases of poisoning of man and lower animals by this plant are on record.

Symptoms: The first symptoms are pain in the bowels, urging to ineffectual attempts to evacuation, burning in the stomach, nausea, vomiting, tetanic convulsions which may be severe, resembling those produced by strychnine, or there may be coma without convulsions.

Apparently the plant is not as poisonous in the summer as in the fall, winter and spring.

Poison Hemlock: This is another very toxic plant. It has been introduced in a few places in Iowa. The flowers are white and the plant has compound leaves exhaling the odor of mice.

Symptoms: In lower animals there is observed a dilation of the pupil, followed by weakness of the limbs, passing into paralysis; labored respiration, frequency of breathing diminished, heart action irregular; death preceded by convulsions. In man there are weakness in the lower extremities, staggering gait; in two hours paralysis of upper and lower extremities and slight convulsions; death occurs in a few hours, usually caused by cessation of respiration.

Black Henbane. The Black Henbane *(Hyoscyamus niger)*

has recently been reported to me from several places in Iowa, one from near Belle Plaine. It is a biennial plant with a dull, yellowish flower and purple veins. Hyoscyamin is obtained from the plant.

Blyth gives the action of *Hyoscyamin* as follows:

Thirty-two mgrs. (½ gr.) begin to act within a quarter of an hour, the face flushes, the pupils dilate, there is no excitement, all muscular motion is enfeebled, and the patient remains quiet for many hours; 64.8 mgrs. would possibly be a fatal dose. The root is more poisonous than the leaves. The seeds of *Datura* contain a considerable quantity of *hyoscyamin;* they are often mistaken for other seeds, such as poppy.

The Jimson weed is one of the very common and unsightly weeds in Iowa, frequently in barnyards. It has a disagreeable odor. The flowers are white, funnel form. I have been informed several times that Jimson weed was permitted to grow in barnyards, and especially the hog lot, because of its value in connection with hog cholera. The plant has valuable medicinal properties, especially in connection with Asthnose, but its use for hog cholera is an idle dream.

Professor Chesnut, in his work on the Poisonous Plants of the United States, referring to the Jimson weed, says:

"The poisonous alkaloids, *atropin* and *hyoscyamin,* the active constituents of belladonna, are found also in both of the Jimson weeds. *Hyoscyamin* is the poison of the henbane, and as it is identical in its physiological action with *atropin,* the above named plants present the same symptoms of poisoning, which must be met in the same manner. The alkaloids exist in all parts of the two daturas. The seeds are especially poisonous."

Cases of poisoning arise in adults from excessive use of a stimulant or a medicine. Children are sometimes tempted to eat the fruit, if they are permitted to play where the weed is to be found. Several cases of this kind were reported to the department during the fall of 1897. At Alpena, Mich., five children were badly poisoned in August by eating the seeds of the purple-flowered species, which was cultivated in a garden as a curiosity under the fanciful trade name of "Night Blooming Cactus." In September a boy was killed in New York by eating the seeds of a Jimson weed which was permitted to grow in a vacant lot; his brother, poisoned at the same time, was saved only with difficulty. In October two other cases occurred in New York. Four children were playing in one of the public parks of the city, where Jimson weeds were growing luxuriantly. The boys imagined themselves Indians and roamed about and ate parts of various plants. Three of them ate the seeds of the Jimson weed. One died in a state of wild delirium; another was saved after heroic treatment with chloral hydrate and morphine; the third, who ate

but few of the seeds, was but little affected. Children are also poisoned by sucking the flower, or playing with it in the mouth. The fresh green leaves and also the root have occasionally been cooked by mistake for other wild edible plants. One or two instances are recorded in which cattle have been poisoned by eating the leaves of young plants which were present in grass hay, but these animals generally either avoid the plants or are very resistant to its poison.

The subject of poisons would not be complete without some reference to the Poison Ivy. This trailing or climbing shrub with leaves of three leaflets climbing by aerial rootlets is widely distributed in Iowa. Some people are extremely sensitive to its action. There are several species. In my Manual of Poisonous Plants I described three species—the swamp or poison oak, occurring in swamps, the California species and the Iowa species.

Poisonous Properties: All three species are poisonous to many persons, some persons being much more sensitive to irritation from the plants than others. Dr. J. C. White describes the effect of poison oak and poison ivy. He had collected freely of the plant for many years without any disturbance. Specimens were picked on September 28th, October 6th and October 10th. He felt a sensation of irritation about the eyes and throat from the specimens of poison oak collected on the first named date while working with the plants under an Argand gas burner, but nothing further was noticed. No unpleasant symptoms were observed from the poison oak *(R. venenata—R. Vernix)* collected October 6th. From that collected October 10th he experienced symptoms similar to those observed September 28th. On October 12th a single vesicle with a peculiar thick cover appeared; the next day another and larger one appeared on the wrist; two others came on the fourth day; others continued to appear up to November 3d, after which date the effervescences gradually subsided and were no longer perceptible. In another case described by Dr. White, the head was greatly swollen and features greatly disturbed. The skin of the face and neck was deeply oedematous and largely covered with vesicles of all sizes, "many of which were seated on an erythematous base, others being still in their papular stage of development." There were also large excoriations from which fluid was exuding freely, which on drying formed small crusts. The hands were also covered. "The subjective symptoms were great retching and burning of the parts affected, with the feeling of local discomfort, consequent upon so great swelling of the features. The eyes were nearly closed. There was a slight general febrile action." Dr. White also reports the death of a child from a severe case of poisoning from poison ivy. A recent case was reported from Packwood, Iowa, where a fourteen-year-old girl died after terrible suffering from

the effects of coming in contact with the ivy, her face alone showing the eruption from the poison.

Taking the simple vesicle, with scarcely any erythema surrounding it or any very perceptible infiltration of the underlying tissues, as the type of the eruption, whether occurring singly or in groups, we may have in a small percentage an abortive attempt at vesiculation, and an arrest of the development at the papular stage—a failure, that is, of the free exudation to force apart the layers of epithelial cells; or a considerable infiltration into the papillary layer may elevate a cluster of the vesicles noticeably above the general surface, or they may be surrounded by a well-defined erythema or congestion of the tissue immediately surrounding them, in consequence mainly of the scratching and itching, which are the only subjective symptoms present.

Sugar of lead is the most popular remedy. No doubt it does some good. The best thing to do is to wash immediately with soap and water and the scrubbing brush. This removes the poisonous substance.

I call attention to the ivy because of its interest to most individuals. I doubt whether stock has ever been poisoned by Poison Ivy. Few people seem to know Poison Ivy.

SURGERY OF NASAL CAVITIES AND SINUSES

DR. H. E. BEMIS, AMES, IOWA

Conditions affecting the nasal cavities and sinuses are classified, as, first, tumors, and under this heading the subhead of inflammatory tumors, specific tumors and congenital tumors.

Then the second main heading, which is hard to separate from the first, and which might in some cases be included under the first, and in other cases might constitute a separate head, is that of cysts. We know that certain kinds of cysts or cystic degenerations occur in the process of tumor formation, and those cysts should practically be included with tumors. But we have other cysts, chiefly the mucous retention cysts which occur in the nostril, and also as a result of the plugging of any of the ducts of the mucous glands. These are on the whole, unimportant.

Then, affecting the nasal cavity, thickening and deflection of the nasal septum; the third main heading. The fourth main heading, surgery for the purpose of drainage of the sinuses; and, fifth, surgery of the sinuses necessary for the repulsion of teeth.

Under the heading of tumors, first, we have the specific tumors such as sarcomas, carcinomas, osteomas and chondromas, and possibly others, but these being the most common.

I have here a specimen from an eight-year-old standard-bred stallion, which was affected with what we had diagnosed to be a carcinoma involving the alveolar cavities of the fifth and sixth upper molars, and spreading by metastasis from that point to the entire cervical chain of lymph glands, and into the lungs, as was shown on post mortem.

It is the usual history in cases of malignant tumors of this kind, that the animals lose flesh rapidly, and the process develops quite rapidly, and is manifested by an outgrowth of either granular or easily broken-down tissue, and in the course of a comparatively short time the teeth become loosened in their cavities and can be removed easily, or possibly drop out.

Of course, it is needless to say that in these cases treatment is contra-indicated, and I think this condition is of great importance to have in mind, so we will not make the mistake of extracting these teeth and giving perhaps an otherwise rather favorable prognosis.

This case (referring to a horse which was at that moment

brought in) has just come into this clinic, so we have not had opportunity to make a positive diagnosis. It does not affect the nasal cavities or sinuses, but may be another case of the same kind just mentioned, or possibly slightly different, but of the same nature. The history of this case is that there was a small enlargement on the mandible proper, opposite about the third or fourth mandibular tooth, two years ago, and thinking that a tooth was involved the veterinarian in charge trephined and repelled the tooth opposite the enlargement, but the tooth repelled was found to be normal. The tumor grew more rapidly, from that time to this, and has now reached the size which you see here. It seems perfectly hard, and osseous, and on curettement of the cavities seems to contain a cancellous bony material. We would conclude from a clinical examination and from the history of the growth, etc., that this is probably an osteoma or possibly an osteo-sarcoma, or carcinoma, although it does not seem to be malignant to any extent, and for that reason I would rather be inclined to think it an osteoma or some of the less malignant tumors, rather than a sarcoma or a carcinoma.

I show this case to illustrate the fact that we may expect to find a percentage of true tumors involving the roots of the teeth, and consequently in the case of upper teeth, involving the nasal cavities or sinuses.

The inflammatory tumors; that is, tumorous growths which are the result of inflammation, occur in the sinuses. So we may expect to find tumorous masses developing in the nasal cavities or sinuses as the result of continued irritation there, such, for instance, as chronic and continued infection of the alveolar cavities.

I think that will illustrate the three classes of tumors: first, the inflammatory tumors; second, specific tumors, and third, congenital tumors.

I might say a word as to the differential diagnosis among these three kinds, and in that regard I would say that to make a diagnosis of inflammatory tumor we should make a careful search for the tracts through which infection may have gained entrance. If we find defective teeth, loose teeth, teeth from which the gums have retracted, then we would probably conclude that infection gained entrance through that tooth, either through the infundibulum, through the enamel plate or through a break between the tooth and the gum, and in any of these three ways, finally reached the alveolar cavity, set up an inflammation there and consequently an inflammatory growth.

To make a diagnosis of a specific tumor, we should consider the history of the case, the rapidity of the development,

the character of the tissue which makes up the tumor, and its tendency to spread by metastasis. The age of the animal and the species would all make some difference.

To make a diagnosis of congenital tumors, therefore, it would really be for the most part a process of elimination. If it is not an inflammatory tumor or specific tumor, and especially if it is found in a young animal, up to one or two or three years of age, with no cause that we could find, then in all probability, from the clinical standpoint, we would have to conclude that we were dealing with a congenital tumor.

The cysts, mucous and retention cysts, are simple of diagnosis and in treatment. These appear as small, tense, well-defined projections from the mucous membranes or skin, non-inflammatory, and need merely to be completely excised, in order to bring about satisfactory results.

Thickening of the nasal septum and the deflection of the nasal septum may come about as the result of chronic inflammation, such as chronic catarrh following distemper, for instance; chronic collections in the sinuses, or may come from blows over the bridge of the nose, with fractures, the septum being involved secondarily. In these cases the respiration is again interferred with, but without any enlargement of the face. Furthermore, the thickened septum can usually be palpated through the end of the nostril, and the thickness arrived at in this way.

The surgery of thickened nasal septa has been rather unsatisfactory. We mentioned last year, and wrote up for the Journal of the American Veterinary Association, an operation for the removal of the nasal septum.

Referring to the surgery for the drainage of sinuses: We have, of course, two sinuses, the maxillary, which is divided into the posterior and anterior compartment by a transverse septum, and each of these compartments again partially divided into a medial inner and a lateral compartment. Then we have the frontal sinus in the frontal region and extending downward to the point at which the fingers when passed from below upward begin to diverge. That you can rely upon as giving you the lower limit of the frontal sinus. The frontal sinus, as you have already seen from Dr. Murphey's work, communicates very freely with the upper portion of the maxillary sinus, through the large fronto-maxillary opening.

To drain the maxillary sinus it is necessary to trephine at a point just posterior to the lower end of the facial crest, and about one inch above the facial crest. That is, in the angle formed by a line drawn perpendicularly to the end of the crest. This will drain the outer compartment of the maxillary sinus. If the posterior compartment of the sinus is affected it

will be necessary to trephine posterior to the septum or drain through the septum from the first trephine opening. If the inner compartment of this same sinus is also affected it will be necessary either to break through the thin plate of bone which supports the infra-orbital canal and drain it to the outside, or pass over the infra-orbital canal and break through the ventral turbinate bone, into the nasal cavity.

To drain the frontal sinus we may use again one of two methods, the first one a trephine opening at the point previously described, and about one and a half inches from the median plane, in the depression between the nasal and maxillary bones.

The second method is to trephine at this same point or anywhere from that point upward to a line drawn across between the inner canthus of the eye and through such trephine openings, break down the dorsal turbinate bone, into the nasal cavity.

There are advantages and disadvantages in both of these. In draining through the outside the disadvantage is the constant discharge over the side of the face. The advantage is that you do not have to break through the very vascular turbinate. On the other hand, the advantage of drainage through the turbinate is that the drainage is through the nose and not over the side of the face.

The surgery of the sinuses for the repulsion of teeth is usually only possible for the repulsion of the fourth, fifth or sixth maxillary teeth. The fourth one is usually in the lower compartment or the anterior division of the maxillary sinus, at the same point which we have been speaking of for drainage. The fifth tooth can be reached at a point about an inch to an inch and a half posterior to that chosen for the fourth one, and the sixth one, which it is very seldom necessary to repel, I think can be most easily repelled by trephining into the frontal sinus at the height of the inner canthus of the eye, and about an inch and a half from the median line, and carrying the punch through the trephine opening, through the fronto-maxillary opening, down upon the root of the tooth. In this way you get a much more direct route and are in much better line with the tooth than if you attempt to trephine the maxillary sinus. The curvature of the sixth tooth in young horses is so great that it is almost impossible to repel.

PHYSICAL EXAMINATON OF RESPIRATORY TRACT AND PNEUMONIAS

DR. D. S. WHITE, OHIO STATE UNIVERSITY

Percussion of the Thorax.

Percussion consists in striking a body to cause it to vibrate and emit sounds. As different bodies will vary in intensity, so will the sound produced vary. If a solid brick wall is struck with a base drumstick the sound emitted is shallow and flat; if a base drum be struck with the stick the sound produced is deep and full.

While thoraces vary in size, wall thickness, etc., from each other, nevertheless, as all of them contain the same organs and the relative size of these organs to the thorax varies but little, the sound produced by percussion is in health fairly constant and characteristic. It is called the pulmonary resonant sound. By training the ear through practice to appreciate the quality of this sound, taking into consideration the differences in size, wall thickness, etc., one can determine when the sound is physiological or normal and when pathological or abnormal.

The thorax emits sounds when struck because it is set in vibration. The sound, however, comes from the air contained in the lungs rather than from the tissues. As in the healthy lung the quantity of air contained is fairly well fixed, varying, of course, with each inspiration and expiration and the intensity of the respirations, the sound produced by percussion will convey to the ear of the examiner approximately the amount of air contained in the lung. Therefore, if the sound emitted has the pulmonary resonant character, we can assume that little or none of the air contained in the lung has been displaced by solid tissues or liquids.

1. Flat Sound: Most of the diseases which affect the organs of the thorax are inflammations. Before completely developed inflammation passes through two stages: (a) The stage of congestion, which means that the blood vessels involved in the inflammatory process are distended and gorged with blood. The congestion stage will affect the sound of percussion but little, at least in animals where the field of percussion is limited as compared with man. (b) The exudative stage. Here the constituents invade and infiltrate the tissues or collect in cavities which it may reach. In the lung the air cells (alveoli) are therefore filled with exudate, masses of liquid and solids which have displaced the air the lung normally contained. If only a very small area is involved in the inflammation or the healthy lung

cover the inflamed area, the normal percussion sound will not be affected. On the other hand, if the inflamed part is large enough (ad minimum, the size of a double clenched fist in large animals) the percussion sound is changed in character, becoming usually shallow, higher pitched, or what is commonly termed *flat.*

Sometimes the inflammation involves thoracic organs other than the lungs. For instance, the exudate may come from the pleura. In this case it is usually mostly fluid, which collects in the thorax with remarkable rapidity, so that in twenty-four hours several gallons may accumulate. The lung floats upon the surface of the fluid provided it is not anchored to the chest wall by some previous inflammation. The effect of the accumulation of exudate extrapulmonary is much the same as when intrapulmonary, each producing a flat sound. If the inflammation involves the heart sac, as is so frequently noted in cattle suffering from serofibrinous pericarditis from direct injury, the percussion sound will also obviously be flat.

In tumor formations or with hydropsical conditions and the rarer degenerative changes which the lungs may undergo, a flat sound on percussion will predominate. Therefore, whenever the normal pulmonary resonant sound is replaced by a flat sound, we know that either solid or fluid tissue has displaced some of the air in the lung. A flat sound *per se* means this and nothing more. To consider it pathognomonic of pneumonia is an error. While flatness is present in pneumonia and forms a very important clinical symptom of this disease, nevertheless the other signs of lung inflammation must be also taken in consideration.

2. Tympanitic Sound: Another abnormal sound emitted by the thorax is the so-called tympanitic sound. The character of this sound is somewhat musical. It may be imitated by opening the mouth and striking the cheeks with the hand. This sound originates from an air cavity surrounded by a rather rigid wall, and in communication with the outside world. In very small animals the pulmonary percussion sound is normally tympanitic. The tympanitic sound is occasionally heard in large animals where a superficial area of air-containing lung is surrounded by a zone of solid exudate. Or where caverns or dilated bronchi exist, provided that between them and the chest wall consolidated lung tissue is present. In pneumo-thorax, where a prolapse of the stomach or bowels has occurred into the thoracic cavity, the percussion sound is tympanitic.

3. Hyper-Resonant Sound: A hyper-resonant, overloud or exaggerated resonant sound occurs where the quantity of air in the lungs exceeds the normal. The character of this sound is somewhat like the tympanitic, but is lower in pitch and fuller. In practice the hyper-resonant sound is met with most commonly

in "heavy" horses suffering from alveolar pulmonary emphysema. It is also heard in pneumo-thorax.

4. Amphoric Sound: This sound resembles somewhat that produced by blowing across the mouth of a bottle. It is a variety of the tympanitic sound. When heard over the thorax it is always pathological. In animals it is rarely heard, but occasionally when a cavern in the lung exists and the thorax over it is thin enough it can be heard. It is also heard in pneumo-thorax.

5. Cracked Pot Sound: This is a pathological sound which may be imitated by shaking between the palms of the hands some coins or by striking the hands loosely clasped against the knee.

The sound of the cracked pot is heard on percussion where a cavity exists in the lung and in pneumo-thorax. Most commonly, however, in practice it is found to occur either where healthy lung tissue lies between the thoracic wall and the area of consolidated lung or where two areas of consolidated lung are separated by a thin area of healthy lung. It also exists where subcutaneous emphysema appears.

Auscultation.

By auscultation as applied to the thorax we mean simply listening over the chest to determine whether or not the sounds of respiration are normal.

The following respiratory sounds are recognized: (a) Vesicular sound; (b) bronchial sound; (c) rales or rhonchi, of which there are several varieties; (d) friction sounds.

1. Vesicular Sounds: The vesicular sound is heard at each inspiration in healthy animals. It is rather soft in character and may be imitated by shaping the lips to pronounce the letter V and at the same time inhaling. The origin of this sound is in dispute. It is probably, however, nothing more than a laryngeal and therefore stenotic tone modified in the smaller bronchi and alveoli of the lung. It is the normal respiratory sound heard over the thorax. In the horse it is much softer than in the ox. Obviously it is intensified by exercise. The presence of this sound tells us that the alveoli are not blocked with fluid or solid tissue, but are free and contain air.

In disease of the lungs or pleura the vesicular sound may be modified:

1. Weak or absent. If a marked stenosis exist in the bronchi. Where the lung tissue has lost its normal elasticity as occurs in alveolar pulmonary emphysema, interstitial emphysema, collapse of the lung following marked protrusion of the diaphragm due to bloating of the stomach or bowels or prolapsus

of the abdominal organs into the thorax. The vesicular murmur may also be weakened when the respirations are very slow and superficial, as occurs in unconscious patients or where they are suffering from pleuritis, peritonitis or pleurodynia, causing the respirations to become very painful. It may also be weak where extensive adhesions occur between the lung and rib pleurae.

Obviously, if the alveoli are filled with exudate, as occurs in pneumonias, or wherever the lungs are extensively consolidated, the vesicular sound is absent.

If the thoracic cavity is filled with fluid or air and the wall greatly thickened the vesicular murmur will be diminished.

Absence of the vesicular sound and all other respiratory sounds (respiratio nulla) occur: In complete obstruction of a principal bronchus (foreign body, worm coil, compression from enlarged lymph glands or tumors). More commonly, however, it follows a consolidation of the lung over a considerable area. It is also a symptom of an accumulation of fluid in the thorax, pneumo-thorax or where the chest wall has become greatly thickened.

2. Exaggerated vesicular murmur. This occurs in all cases of dyspnea, provided the alveoli are free. Otherwise, if a part of the lung is consolidated, the vesicular murmur will be louder in the still healthy lung tissue surrounding. In this instance it is compensatory. The vesicular sound may be augmented by other respiratory sounds. For instance, if the laryngeal sound is abnormally louder or if the bronchial mucosa is swollen or exudate or membranous deposits appear on its surface, producing stenosis.

3. Sacculated vesicular respirations. These are not uncommonly heard in perfectly healthy animals, but they also occur accompanying painful diseases (pleuritis). Occasionally they may accompany bronchitis, masses of exudate blocking the lumen of some of the bronchi.

2. Bronchial Sounds: The bronchial sound resembles that sound which emanates from the larynx or trachea. It may be imitated by arranging the vocal organs to pronounce the guttural German "ch" and exhaling forcibly. It is probably a continuation of the laryngeal sound modified in the bronchi.

Physiologically the bronchial tone is heard over the larynx and trachea, especially in the ox. In the thorax it is never heard in healthy horses. In the ox in the region of the 3-4 intercostal a soft and weak bronchial sound is recognizable. The leg must be drawn well forward to expose this area of auscultation. In fat animals it may be missed.

In the horse, when it is heard over the thorax, it is always due to some pathological condition.

Obviously, the air passing in and out of the bronchi will produce sound as it does in the larynx and trachea. However, as the bronchi are surrounded by air containing lung tissue, a bad conductor of sound, it cannot be heard when the ear is placed to the chest. There are two conditions which make the bronchial sound audible on auscultation. 1. Where there exists a stenosis of the larynx and trachea, large bronchial branches or nasal cavities. In large animals, however, the bronchial sound is heard usually no farther back than the 8-10 rib. In bronchial stenosis it is usually heard only on one side. 2. When the air spaces in the lungs become filled with fluid or solid exudate. In the latter case two factors bring out the bronchial sound: (a) The dyspnea, and (b) the fact that a better medium now exists between the ear and the bronchi for the transmission of the sound.

3. Rales: Rales or rhonchi are sounds emanating from the bronchi. They originate in a variety of ways, but in general come from liquid, semi-liquid or solid masses of exudate which accumulate in the bronchi. Depending upon the character of the sound, which in turn depends upon the origin, the following rhonchi are distinguished: 1. Moist rales. 2. Crepitant rales. 3. Dry or sibilant rales.

If the exudate is fluid the air passing through it will cause it to bubble and foam, producing a rattling, boiling sound which is spoken of as a moist rale.

Peculiarities of Rales: In judging rales we should take into consideration the time they appear, their size, intensity and tone.

From the standpoint of occurrence they may be inspiratory, expiratory, interrupted or continuous. Not unrarely rales are first heard after coughing or moving it over rough ground for a distance of fifty feet or more. In other words, anything which makes the respirations more intensive, like coughing or exercise, will bring out the rale more prominently. In seventy-seven cases of open pulmonary tuberculosis in living cattle Ostertag determined rales in only 17 per cent when the cattle were at rest, but after exercise at a trot rales were heard in 79 per cent. On the other hand, feeble rales may be made to disappear by severe exercise. The injection of pilocarpin or arecolin can produce rales in perfectly healthy animals. The strength (loudness) of a rale depends upon the viscosity of the exudate, the amount of air passing through the bronchi and the distance from the surface. Obviously, the larger the bronchus, the more fluid it contains and the nearer it is to the ear the louder will be the rale.

The tone of the rale depends largely upon the consistency of the exudate. The tougher and less fluid it is, the sharper and drier will be the rale.

Diagnostic Importance of Moist Rales: The presence of

moist rales on auscultation over the thorax speaks for the presence of fluid masses of exudate or other fluids in the bronchi which are still free to admit air. Therefore, they occur in edema of the lung, bronchial catarrh, where pneumonic exudate has become fluid (stage of resolution in pneumonia), ichoric masses in putrid bronchial catarrh or gangrene of the lung. They may also come from blood in pulmonary hemorrhage or saliva or water which has been swallowed "the wrong way." The location of the origin of moist rales is not always easy. It must be remembered that rales which emanate from the trachea or larger bronchi can also be heard in the chest, although the sound is modified as compared with the sound emanating from the seat of their origin.

Rales are fine, crackling sounds resembling the sound produced by throwing salt on a hot plate. They may be imitated by rubbing a lock of hair between the fingers or by moistening the finger tips, alternately pressing them together and pulling them apart.

Crepitant rales originate in the alveoli or bronchi.

Diagnostic Importance of Crepitant Rales: They occur in pulmonary edema, the congestion stage of fibrinous pneumonia, and very rarely in broncho-pneumonia or chronic pneumonia. Very fine moist rales may sound a great deal like them, but usually crepitant rales occur at the acme of inspiration, while fine moist rales are more apt to appear at expiration.

A crepitant rale of special character is heard in pulmonary interstitial emphysema due to the movement of the air bubbles in the connective tissue of the lung. The sound is more cracking or crunching, resembling somewhat the sound produced by walking over frozen snow.

Dry rales are piping, squeaking, whistling, sibilant sounds.

Origin: They originate from projecting, solid or semi-solid masses of exudate on the bronchial mucosa set in vibration by the passing air. In general there are two sorts, viz.: A fine whistling rale which originates in the smaller bronchi, and a large or sonorous rale with its seat in the larger tubes.

They speak for bronchitis and are therefore heard in chronic bronchial catarrh, in alveolar pulmonary emphysema, tuberculosis, lung worm pneumonia, and in chronic pneumonias generally. In eighty-three cattle suffering with tuberculosis, Ostertag found in fifty-five dry, two moist and seven mixed rales.

4. Friction Sounds: These sounds resemble that produced by rubbing the concha of your ear with a finger.

The sound is most commonly heard in the lower part of the thorax behind the muscles of the shoulder. This is due to the fact that pleuritis is most apt to occur in this region and the lung movement here is greatest at inspiration. More rarely the sound

is heard in the upper thorax. In the neighborhood of the first four intercostal spaces pleuritic friction does not occur, as the lung movement in this region is more radial and limited along the thoracic wall. However, if the heart's sac is involved it may occur.

The friction sound can usually be heard best at inspiration; it rarely is audible during both phases of respiration and is very rarely heard exclusively at expiration.

Friction sounds may be confused with fine rales, sharp vesicular sound, pericardial bruits and various extraneous sounds. Contrary to friction sounds, rales are heard usually over most of the chest, are influenced by coughing and breathing (may disappear or become modified) and are not affected by pressure on the intercostal spaces. The sharp, vesicular sound is continuous, no sensitiveness is present on palpation of the intercostal spaces and it is not affected by cough artificially induced.

Diagnostic Importance of Friction Sounds: Usually due to pleuritis. It will be heard only in the first (dry) stage and usually disappears entirely in twelve hours to three days. It may, however, continue after the effusion has taken place, but can be heard only above the surface of the fluid. After the absorption of the exudate the friction sound may re-occur.

In pleural tuberculosis or in pleural tumors (sarcomatosis, carcinomatosis) the friction sound is rarely heard, as the surface of these growths is usually too smooth. However, if an acute pleuritis co-exist or calcarious degeneration has taken place the sound becomes heard.

CLINICAL CASES.

Case No. 675. Gray Mule: Mule became lame at pasture, August 20, 1916, but was not examined until August 25th, when a pus discharge was noticed at its heel, also a puncture wound at middle of frog. Animal very lame; respiration, pulse and temperature normal. Examination made by Dr. Adams.

Diagnosis: Fracture of third phalanx.

Treatment: Splint dressing, leaving drainage below. Within a few days the dressing became soaked with discharges and was removed; more complete drainage was established through digital cushion and foot dressed daily. Case discharged October 4, 1916. Process nearly healed.

Case No. 680. Gray Mare, Five Years Old: Swelling appeared on lower right mandible about two years ago. Trephined in May, 1916, for removal of tooth; since that time the enlargement doubled in size.

Symptoms: Firm, smooth, painless. Enlargement of mandible half the size of a man's head. Teeth and alveoli not involved.

Diagnosis: Osteosarcoma.

Treatment: Not advised.

Case No. 682. Brown Mare, Six Years Old: Firm, painless, slowly developing enlargement of mandible; no discharge and apparently no loosening of the teeth or interference with mastication.

Diagnosis: Osteosarcoma.

Treatment: Not advised.

Case No. 686. Bay Gelding, Five Years Old: Animal had distemper during the winter and several abscesses formed in subparotid region. Horse first noticed to be off in wind about April 15, 1916, and developed into chiefly an inspiratory breathing with some snoring sounds. Condition became progressively worse.

Diagnosis: Laryngeal Hemiplegia and Chronic Laryngitis.

Treatment: Operation by Dr. Bemis.

Animal became worse and was kept at the college. Post mortem showed a severe chondritis involving the cricoid cartilage and the arytenoid cartilages in such a way as to narrow the lumen of the larynx and render the animal useless.

Case No. 693. Sorrel Mare, Eight Years Old: History of having had distemper.

Symptoms: Inspiratory dyspnea increased by exercise.

Diagnosis: Laryngeal Hemiplegia.

Treatment: Operation by Dr. Adams.

Case did well and was discharged October 5, 1916.

Case No. 694. Bay Mare: No reliable history and animal sent in with tube in trachea.

Symptoms: Inspiratory Dyspnea.

Diagnosis: Laryngeal Hemiplegia.

Treatment: Operation by Dr. Adams, September 6, 1916.

Animal discharged in good condition September 27, 1916.

Case No. 697. Bay Gelding, Four Years Old: History.—Animal apparently suffered from forage poisoning two years ago; has been affected with incoordination of the hind limbs since. Left fore limb raised high in walking; left hind limb carried in adduction, which resulted in interfering and the production of a large callous on right fetlock. Rectal examination revealed nothing abnormal.

Post Mortem: No fractures or deformity could be found. The lumbar portion of the spinal cord was soft and degenerated, which probably accounts for the paralysis.

Case No. 720. Sorrel Mare, Eight Years Old: Animal had been affected with inspiratory dyspnea for some time. Sound, typical of roaring.

Diagnosis: Laryngeal Hemiplegia.

Treatment: Both Ventricles stripped by Dr. Adams September 8, 1916.

Animal discharged in good condition October 6, 1916.

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